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NEW CONCEPTS IN CORRELATION OF SURGICAL ANATOMY AND EMBRYOLOGY IN LATERAL CERVICAL VESTIGEAL STRUCTURES.*†

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The embryology of the cervical region is fascinating and complicated. We may recall from our student embryology the phrase, ontogeny recapitulates phylogeny, or the embryological history of the individual organism recapitulates the history of the race. The branchial arch structures represent that phase in the evolution of man wherein his forbears existed in an aquatic environment. The word branchial is defined as pertaining to or resembling the gills of a fish. Frazer[1] has several objections to the term branchial in describing this phase of development in man, especially since at no time are definitive branchial or gill structures formed in the recapitulation process. The entire arch phase exists as a visible external structure for only three weeks. Congdon[2] stated that the arches do not in any complete sense recapitulate their phylogenetic history, and that the process is much foreshortened and distorted.

Most authors presenting a case of lateral cervical fistula stress the importance of embryology in understanding its surgical anatomy. In attempting a correlation there are several points which tend to confuse. The author was con-

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fronted with a case of complete lateral cervical fistula which opened internally into the tonsil fossa, then passed beneath the posterior belly of the digastric muscle, coursed anterior to the region of the carotids, and opened externally near the sternocleidomastoid attachment to the clavicle. According to older writings this would appear to be a fistula of the second pharyngeal pouch breaking through the closing membrane into the second branchial groove and emerging externally through the duct of the precervical sinus. Wenglowski,[3] however, maintained that the hyoid bone marked the inferior level of the branchial arch region on the post-embryonic neck and, because complete fistulae often open much lower, he presented his thymic duct theory in 1913, and a wide measure of acceptance was recorded. The thymic duct itself, however, is a third pharyngeal pouch derivative, and according to embryologists the internal opening should be in the pyriform fossa and it should not pass between the internal and external carotids as the majority of complete fistulae do. To add to the confusion, second pouch fistulae should course between the internal and external carotids and not anterior to both, as in the author's and other reported cases.

A historical summary by custom should be inserted here, but it will be entered later so that it may be better appreciated. By organizing the literature, correlating the anomaly with this literature, and with some arm-chair hypothesizing a tenable concept can be evolved in explanation of the mechanism of development of lateral cervical vestigeal structures which does not violate accepted embryological concepts. While this theory is not intended to represent the complete answer, it is at least a reasonable correlation of surgical anatomy and embryology; the pieces of the puzzle fit together to make a whole picture, but whether it is "the" whole picture remains to be seen.

THE BRANCHIAL ARCHES.

Before proceeding it would be well to orient ourselves topographically with regard to the arch area on the side of the head. As can be seen in Fig. 1, before the neck develops in

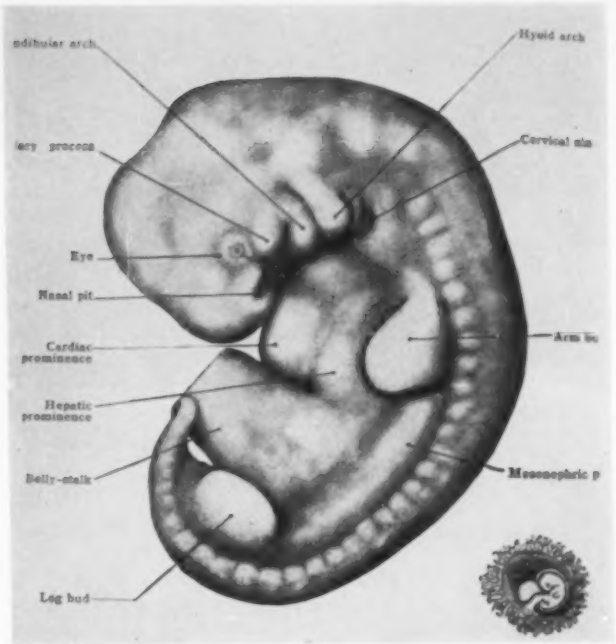

Fig. 1. (Taken from Patten.[9] by courtesy of the Carnegie Institution of Washington): A. Human embryo late in the fifth week. Length 6.5 mm.; retouched photo (x15) of embryo 6592 in the Carnegie Collection. Small sketch, lower right, shows actual size of embryo.

the post-branchial phase, the head is curled directly over the pericardium. Frazer[4] ascribes this to the fact that the dorsal structures of the head at first grow more rapidly than the ventral structures. As the neck develops, the ventral area grows more rapidly, and the head is lifted from the pericardium. The arch field lies initially from before backwards with each arch disposed in a superior-inferior direction. As the neck develops a ventral arc is described due to the rapid ventral expansion, and the structures derived from the arches as the mandible and subjacent transverse flexure line of the neck are disposed in an anterior-posterior direction. For this reason the terms dorsal and ventral as opposed to superior

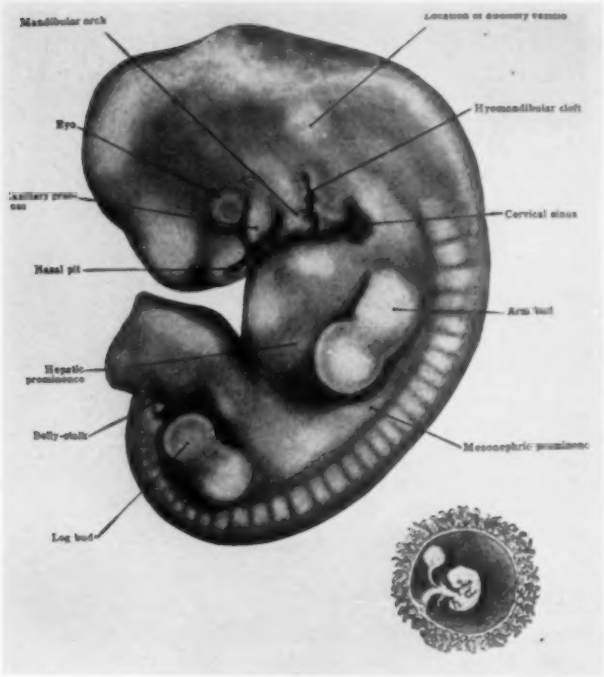

Fig. 1. B. About the middle of the sixth week. Length 10 mm.; note cervical sinus further recessed and prominence of first and second arches with hyomandibular cleft or first groove.

and inferior, and cephalad and caudal rather than anterior and posterior will be used.

The arches appear externally from the fourth through the sixth weeks (embryo lengths 4 mm. to 13 mm.). Only four arches appear externally in man; the fifth and sixth arches not being represented on the surface. The arches consist of mesodermal bars covered by ectoderm externally and entoderm internally. Caudal to each bar, and numbered according to this bar, are the branchial grooves (clefts or furrows); here the ectoderm and entoderm approach each other, with the mesoderm in a relatively thin layer between them. The

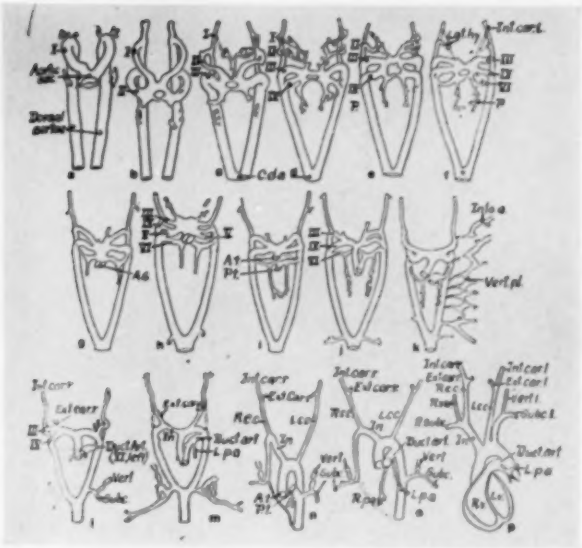

Fig. 2. Ventral views of aortic arch system in a series of human embryos; in the earliest stage only the first two arches are present. The lengths of the respective embryos, from which these diagrams were made are as follows: a, b, 3 mm.; c, d, 4 mm.; e, 5 mm.; f, 6 mm.; g, 7 mm.; h, 7 mm.; i, 8 mm.; j, k, 11 mm.; l, 13 mm.; m, 14 mm.; n, 16 mm.; o, 18 mm.; p, full term fetus.

I to VI—aortic arches; c. d .a., common dorsal aorta; int. car., r. and l., right and left internal carotid arteries; ext. car., external carotid arteries; In., innominate artery; vert., vertebral artery; subc., subclavian artery; A. t., aortic trunk.

Note: In g, aortic arches I and II have completely disappeared. In l, external carotid artery appears from mid-portion of aortic arch III. In m, the segment of dorsal aorta between aortic arches III and IV is disappearing, to form the definitive common and internal carotid arteries.—(From Congdon,[2] taken from Jordan and Kindred, Textbook of Embryology, 5th Ed., D. Appleton-Century Co., 1948).

entodermal evagination corresponding to each ectodermal groove is termed a pharyngeal pouch. These areas of contact or near contact are termed closing membranes and correspond to the gill membranes of aquatic animals.

Some arch structures are evident before the arches are externally evident, *e.g.*, Congdon[2] reports the aorta of the first arch is present in a 1.3 mm., or 19-day-old embryo. Similarly, Frazer[4] reports that at the time the visible manifestation of

Fig. 3. Lateral view of aortic arches of left side of an 11 mm. human embryo. The "x" was added by the author to mark the approximate site at which the external carotid artery will come off at the end of the arch phase (13 mm. embryo).

Note: Any third pouch derivative, whether branchiogenic fistula or thymic duct vestige, is related caudal to the common-internal carotid axis, derived from aortic arch III. (From Congdon,[2] taken from Jordan and Kindred, Textbook of Embryology, 5th Ed., D. Appeton-Century Co., 1948).

the third arch externally is fading, the deeper structures associated with the arch have either disappeared or migrated.

This process of appearance and disappearance, migration or non-migration, results in many controversies in regard to anomalies and their variation from case to case. Several instances of this, in which earlier formed structures migrate or disappear before certain later structures appear, will be touched on in this paper. For example, the stylopharyngeus muscle, a third arch derivative, migrates cephalad into the domain of the second pouch, and if an anomalous fistula develops from the second pouch, depending on the exact time of its development in relation to the migration of the third

Fig. 4. Lateral X-ray, author's case, with lipiodal filled tract. Note its course, anterior to the carotid sheath, and just dorsal to the lesser cornu of the hyoid.

For roentgenograms of complete fistulae surgically proven to have passed between the internal and external carotids, and for a roentgenogram of a third pouch origin incomplete internal fistula, suggesting a course dorsal to the carotid axis (not surgically confirmed), see: Parsons, Philip B., Lateral Cervical Sinus; Report of Three Cases with Brief Review of the Literature, Amer. Jour. Roentgenol., 67:757-762, May, 1952.

arch structures including the stylopharyngeus, the fistula may, and has, passed beneath, over or even through the muscle. Much heated debate has ensued, the basis of argument revolving around the concept that if the anomalous fistula passed caudal to the third arch structure, the stylopharyngeus, the fistula should be of third arch origin. As

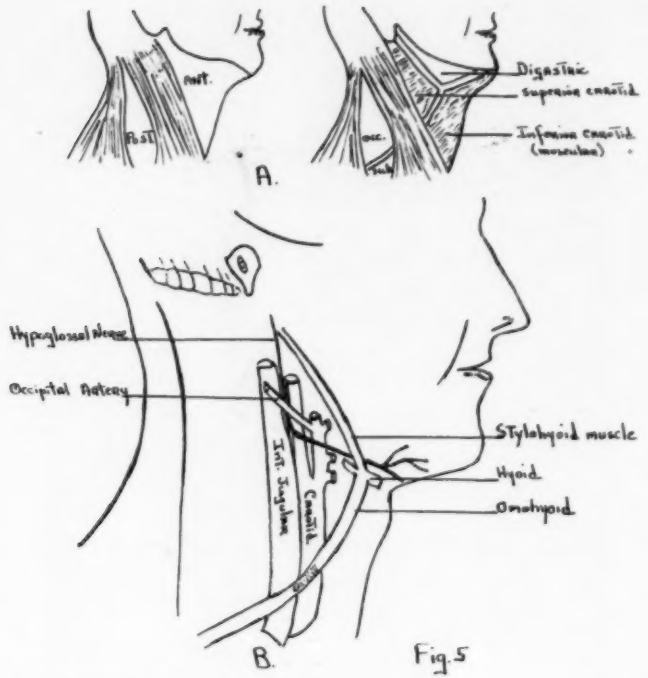

Fig. 5

A Neck Triangles
Approximate area of superior and inferior carotid Triangles combined in Frazer's Third each area.

B. Carotid Axis-Hyoid Relations
Hypoglossal Nerve-Sternomastoid Artery Relations
The nerve may hook behind either the occipital artery or its sternomastoid branch. In any event, the peripheral passing sternomastoid branch always lies ventral to the nerve.

will be brought out in this paper, the more pertinent criteria are the relation to vessels and nerves, and the site of internal opening for a fistula; relations to muscles, as with the stylopharyngeus, is not as decisive. In the same manner it will be explained in more detail that whether a second pouch fistula passes between the carotid arteries or anterior or ventral to both, is a result of the developmental time lag,

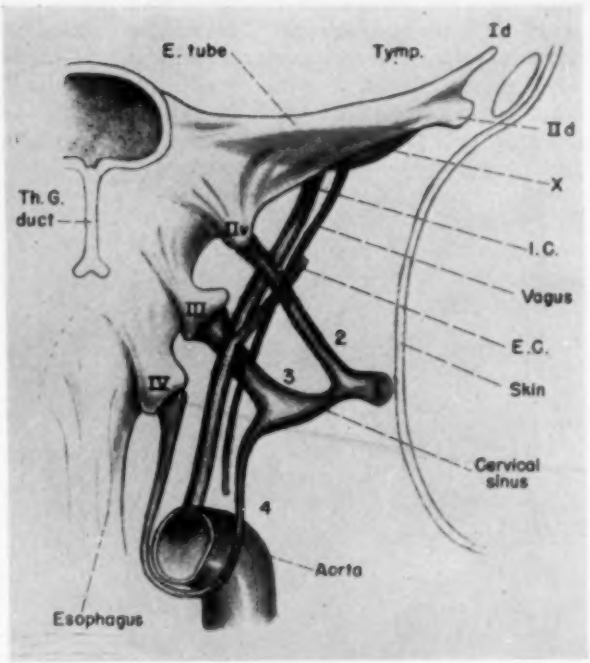

Fig. 6. Schematic figure to show the vestigeal structures in the neck, with their relations to main arteries and nerves. I-d, II-d, dorsal angles of first and second lateral pouches; II-v, III, IV, "internal pharyngeal ducts" derived from ventral angles of second, third and fourth lateral pouches; 2, 3, 4, "external pharyngeal ducts" derived from second, third and fourth external grooves; X, layer of entodermal cells cut off from lower part of Eustachian tube; E. Eustachian tube; E. C., external carotid; I. C., internal carotid; Th. G. Duct, thyroglossal duct. Note: Junction points of II-v, III and IV with 2, 3, and 4, respectively, are the closing membranes.—(As redrawn by Proctor[21] from Frazer[1]).

coupled with the appearance and disappearance of major vascular structures in the branchial arch area.

Each arch develops an aorta, a cartilage and muscle tissue and associated with each, but not developed initially within it, is a cranial nerve. The Vth, VIIth, IXth and Xth nerves are associated respectively with the first, second, third, and fourth and fifth arches; the superior laryngeal division of the Xth nerve, with the fourth arch, and the inferior laryn-

geal with the fifth arch. The primitive arch aortae will be discussed in a separate section. The cartilages develop into adult bony and ligamentous structures including the mandible, ossicles of the middle ear, the stylohyoid ligament, the hyoid bone and the laryngeal and thyroid cartilages. The lesser cornu of the hyoid is derived from the second branchial arch,

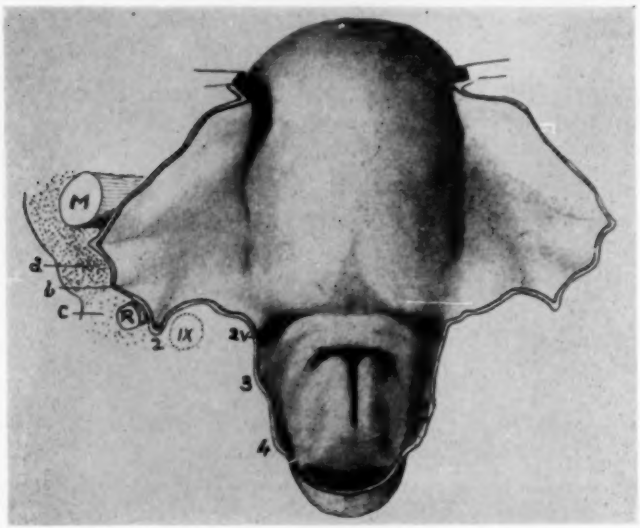

Fig. 7. Pharyngeal floor in 16 mm. embryo. Tubo-tympanic recess, marked off by a groove from the general pharyngeal cavity. M, R, Meckel's and Reichert's bars; IX, glossopharyngeal nerve; a, condensation of handle of malleus; b, groove produced by this; c, mesoderm of second arch; 2, dorsal and lateral part of second lateral pouch; 2-V, 3 and 4, levels of ventral ends of the corresponding lateral pouches. Note: the second pouch extends from the lateral extremity of the tubo-tympanic recess to the base of the tongue, from 2 to 2-V (cf. 2-V to 2 with II-v to II-d, Fig. 6).—(From Frazer[7]).

as are the stapes, styloid process and stylohyoid ligament. An elongated styloid process may actually join with the lesser cornu of the hyoid. The body of the hyoid is derived from the third arch; therefore, congenital fistulae or other anomalous branchial vestiges of second pouch and groove derivation will of necessity course above the body of the hyoid and dorsal to the lesser cornu (see Fig. 4), while third pouch derivatives

will of necessity pass caudal, or beneath the body of the hyoid, or through the thyro-hyoid membrane (see Fig. 9). The muscles developed in the arches in some instances migrate a considerable distance, and their origin is identified by their innervation, *e.g.*, the occipito-frontalis is considered a second arch derivative, as it is innervated by the nerve associated with the second or hyoid arch, the facial or VIIth nerve. The sternocleido-mastoid, tongue and infrahyoid muscles are thought to migrate from the occipital and upper cervical myotomes, as nerves from these regions innervate them. The tonsil fossa is bounded by the palatoglossus and palatopharyn-

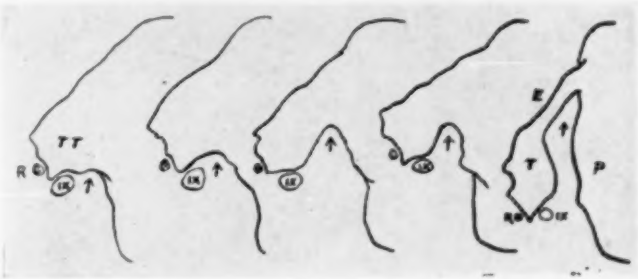

Fig. 8. Outlines of left half of pharynx and tubo-tympanic recess (T T) in embryos of 16, 18, 22, 26 and 35 mm. seen from above, to show the forward growth of the third arch (arrow) passing between the tympanum (T) and the pharynx (P). R, Reichert's bar; IX, glossopharyngeal nerve; E, Eustachian tube.—(From Frazer[7]).

geus muscles, which are variously identified with the Xth and XIth nerves (via the pharyngeal plexus) and so cannot be definitely categorized as to arch of origin, if of arch origin. In general, it can be stated that muscles are not always completely reliable guides in the study of branchial anomalies. As Frazer points out, the most reliable relationship is to vessels and nerves, providing the vessels themselves are developed normally. Wenglowski,[3] in his attempt to prove complete lateral fistulae were of thymic duct origin, claimed that fistulae opening in the region of the tonsil fossa, did not open into the fossa proper but were in relation to the palatoglossus, which he stated was innervated by the IXth nerve, and so claimed this relationship indicated third pouch origin;

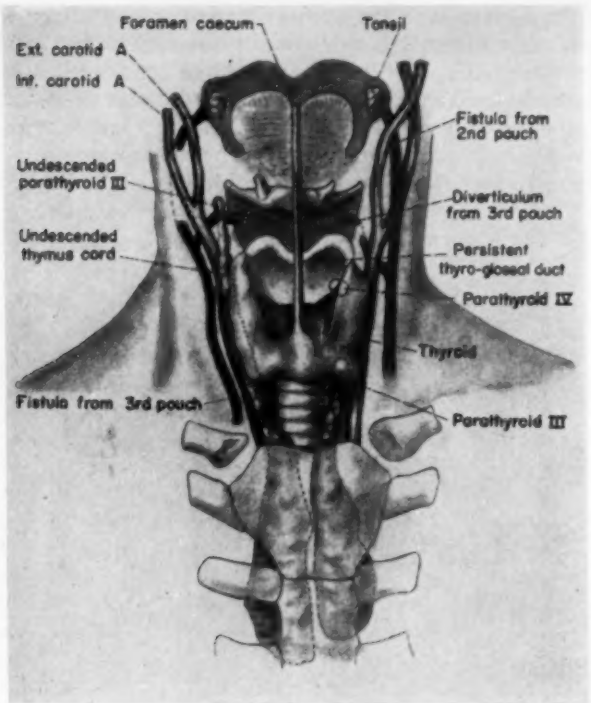

Fig. 9. Schematic diagram from Braus: Anatomie des Menschen, Bd. 2, as redrawn by Proctor,[21] in which is collected a number of anomalous conditions. On the subject's right, note undescended parathyroid III, residual thymus cord, and a branchial fistula from pharyngeal pouch III. A previous connection of the fistula with a diverticulum of the supratonsillar fossa is broken. Center: persistent thyroglossal duct. On the subject's left: note a diverticulum of pouch III, an incompletely descended thymus still attached to parathyroid III, and a fistula of second pouch origin.

Note: The presentation on both sides of third pouch fistulae emanating from the thyro-hyoid membrane, passing dorsal to the common carotid and then lateral to the great vessels, however, the presentation of the thymus cord passing medial and ventral to the carotid is not embryologically sound. See Fig. 3, and Frazer's Manual of Embryology.[7]

in fact, as will be discussed more fully later, fistulae do open into the tonsil fossa proper, and also in relationship to the posterior pillar, and even to Rosenmuller's fossa.

THE BRANCHIAL OR CLOSING MEMBRANES.

In aquatic vertebrates the ectodermal and entodermal tissues of the grooves and pouches make direct contact to form the branchial or closing membranes, which break through normally to form the gill slits and subserve thus a respiratory function. Only in the first groove and pouch in man is direct and measurable contact made which persists post-embryonally as the tympanic membrane. Even here, in line with the distorted recapitulation mentioned in the introduction, Hammar (1902) and Wenglowski[3] report that in human embryos the first pouch and cleft cross each other in "X"-fashion so that while contact is constant it is limited in area to this point of crossing. According to Wenglowski, even the mesodermal bar of the first arch is twisted so that the pharyngeal aspect of the arch lies crosswise to the outer aspect. In the pouch and cleft relationships caudal to the first there is similar distortion, the only consistency being that there is some area of contact between the second pouch and cleft to allow for closing membrane formation, though not always at the same level. Hammar, quoted by Wenglowski, found in a 5 mm. human embryo that the area of contact was with the ventral horn or extension of the second pouch, while Wenglowski, in a 6.5 mm. embryo, found the area of contact with the dorsal extension. For the third and fourth pouches and clefts the relations are more inconstant, with closing membrane formation sometimes occurring for the third, and even less likely for the fourth. In Hammar's embryo the third and fourth pouches extended into the mesoderm unrelated to the grooves. In a 6.5 mm. embryo, Wenglowski found the greater part of a short third pouch taken up in forming a closing membrane. In an 8 mm. embryo, there was variation between the two sides: on one side there was a small closing membrane, while on the other side there was a thick layer of mesoderm separating the pouch and cleft. Piersol (1888), quoted by Hyndman and Light,[5] and Wenglowski, concluded from a study of rabbit

embryos that a breakthrough could occur only via the second closing membrane. Carp and Stout[6] refer to Rabl's findings, that the second groove is deeper and persists longer, and so is the logical point at which a fistula could enter the pharynx. They refer also to the progressively thicker layer of mesoderm between the grooves and pouches in the more caudal structures. The third and fourth pouches are primarily concerned with the development of the thyroid, parathyroid and thymus.

THE AORTIC ARCHES—ORIGIN OF THE EXTERNAL CAROTID ARTERY.

The most recent and comprehensive study of the development and modification of the aortic arches of the human embryo is that of Congdon[2] (1922). The aortic sac or bulbus arteriosus, the heart primordium, communicates via the vessels of the branchial arches, termed the arch aortae, with the paired dorsal aortae (see Fig. 2). Development of the arch vessels is accomplished by the fusion of dorsally growing sprouts from the aortic sac, with those growing ventrically from the dorsal aortae. The first and second aortic arches disappear during the time the third pair of arches is forming (see Fig. 2, a-f). A mandibular artery grows into the region formerly occupied by the first aortic arch, and a stapedial artery replaces the second aortic arch in the second or hyoid branchial arch. These secondarily formed arteries are branches of the dorsal aorta, and have no continuity with the aortic sac. It has apparently been assumed that the arch aortae of the second arch develop into the external carotids. Obviously this is not so. The arch aorta of the third arch is the most cephalad one, which persists as the primordium of an adult vessel. The third aortic arches persist as the roots of the internal carotid arteries and from their anterior surfaces, just about the end of the branchial arch phase in point of time, there arise the external carotid arteries (see Fig. 2, l); hence, the base of each third arch between the external carotid and the aortic sac becomes the common carotid artery (see Fig. 2, n).

To repeat for emphasis, the aortic arches of the second branchial arches disappear and take no part in the formation of the external carotid arteries which first appear in de-

finitive form at the end of the arch phase from the mid-arch portion of the third arch aortae. The dorsal aortae cephalad to the third arch continue as the internal carotids, and give rise to the ophthalmic and middle cerebral arteries. Eventually the segments of dorsal aortae between the third and fourth arches disappear, to form the definitive internal carotids (see Fig. 2, m). It must be assumed that a breakthrough from the second pouch to the second cleft, to form a congenital fistula, occurs in the arch phase, before the definitive external carotids are even formed. In Fig. 3 is Congdon's drawing of the lateral view of the aortic arches of the left side of an 11 mm. human embryo, in the latter phase of the arch system, but before the definitive external carotids are formed. I have taken the liberty of marking the approximate point of origin of the external carotid with "x." It is quite apparent that whether a fistula passes between the external and internal carotids, or ventral to both vessels, depends upon whether the external carotid links up with its cephalad course dorsal or ventral to the second pouch. In several clinical descriptions the fistula courses between the carotids, and in the cases of Dowd,[15] Kleinert[16] and the author, the course is ventral to both. The surgical significance lies in the fact that where the course is anterior or ventral to the carotids the surgery is rendered simple. This relationship can be determined preoperatively by an X-ray study with the fistulous tract filled with a radio-opaque material. It will be seen from the lateral X-ray (see Fig. 4) that the tract crosses the hyoid almost at the lesser cornu, and lies far anterior to the usual course of the carotid sheath. Where the tract crosses the hyoid more posteriorly, and is in topographical relation to the carotid sheath, it can be expected that the course will lie between the external and internal carotids, and the surgery rendered more difficult. It also follows from Figs. 3 and 6, that a third pouch derivative would have to course dorsal to the third arch vessels, the common, and, or internal carotids.

Wenglowski admittedly could not follow the complex development of the arch vessels, and ignored these vessels in relation to the development of fistulae; he felt that muscle relations were more significant.

FATE OF THE PHARYNGEAL POUCHES—THE THYMUS.

The foregut is a prolongation of the primitive intestinal canal. There is no coelomic channel on either side of the gut at this level; the entoderm being separated from the ectoderm by solid mesoderm; thus, with general growth of the region the primitive pharynx increases in breadth. Condensations in the lateral mesoderm cause thickenings to form the arches. The lateral portions of the widened pharynx expand in a dorso-ventral plane to form the lateral pharyngeal pouches between the indentations caused by the arches. The lateral pouches develop dorsal and ventral portions or angles. The ventral angle of the first pouch is obliterated. The dorsal angle, in conjunction with the dorsal angle of the second pouch, forms the tubo-tympanic recess (see Figs. 6, 7).

In the case of the second lateral pouch the distance between the angles increases rapidly. The dorsal angle is incorporated into the tubo-tympanic recess from which are formed the Eustachian tube and middle ear space. The ventral angle extends from the Eustachian orifice to and including the tonsil fossa region. According to Frazer,[7] the separation of the dorsal and ventral angles takes place in this manner: as the tubo-tympanic recess is formed, the second arch lies ventral to the recess, the first arch comes to form the sloping cephalad wall and the third arch the caudal wall. The second arch growth is most effective externally, and it is a prominent structure externally with its groove forming the transverse flexure line of the post-embryonic neck. The third arch, on the other hand, has its most effective growth internally. Its internal bulge first delineates the tubo-tympanic recess caudally. By continued growth of the third arch it approaches the first arch cephalad, thus narrowing the tubo-tympanic recess medially to form the definitive Eustachian tube and separating the dorsal and ventral angles of the second lateral pouch (see Fig. 8). The ventral angle of the second pouch can then be seen to occupy the area laterally from the Eustachian orifice to the base of the tongue. External to this entodermal expansion of second pouch origin is the third arch mesoderm. From this ventral angle, entodermal ingrowths into the mesoderm form the tonsil crypts. This

takes place, however, long after the arch phase has passed, that is, in the fourth month. The crypts form in the lower portion of the angle. The upper part hangs over this ingrowth as the supratonsillar fossa. Lymphoid tissue infiltration takes place even later.

In Wenglowski's time there was considerable controversy over the relation of fistulous openings to the stylopharyngeus muscle, which is innervated by the IXth nerve which courses just deep to it. Being innervated by the IXth nerve, it is a third arch derivative. As described above, however, the third arch mesoderm secondarily migrates into this area during the arch phase of development. It would seem, therefore, that a fistula opening into the pharynx in this region emanates from second pouch area, and its relation to the stylopharyngeus muscle is accidental and of secondary importance in determining the origin of the fistula (see p. 4, section on Arches). It would appear that a fistula opening anywhere from and including the tonsil fossa to the region of the Eustachian orifice is consistent with second pouch origin providing, of course, vascular and nerve relations are consistent with this. Various sites of fistula opening mentioned in the literature are the supratonsillar fossa, Rosenmuller's fossa, posterior pillar, tonsil fossa proper and even just below the tonsil fossa. In the author's case, dye injected into the tract preoperatively appeared in a large upper pole crypt. Taylor and Burwell[8] (1954) reported several cysts as residuals of the second pouch, which extended from the tonsil fossa to the region of the Eustachian orifice. In two of these cases the cysts were bilateral, minimizing any contention that these might be ordinary retention cysts.

The third pouch, like the second, develops dorsal and ventral angles. The internal opening of a vestige of the third pouch would have to be related to the pyriform fossa, according to embryologists. Its relationship to the carotid axis should be caudal. If there were a closing membrane breakthrough with a complete fistula, it should open externally similar to a second membrane fistula. The only difference from a second pouch complete fistula then would be in the level of the internal opening and the carotid axis relationship. There is no case on

record having the relations outlined above for a complete third pouch fistula.

From the dorsal angle of the third pouch develops that portion of the parathyroid gland not derived from the fourth pouch, or parathyroid III. From the ventral portion develops the thymus in the fifth or sixth week. There is little actual descent involved, as it lies almost at once in relation with the pericardium to the lateral side of the great vessels. The distal end of the duct enlarges to form the definitive thymus, while as growth goes on the stalk, elongates and then loses its pharyngeal connection and its lumen is obliterated. The duct, by necessity, if it is of third pouch origin, has to emerge caudal or dorsal to the common-internal carotid axis, the artery of the third arch (see Figs. 3, 6). This is the same relationship which would have to occur for an anomalous branchiogenic structure as a fistula. Frazer[7] describes the course of the thymic duct as given above, emerging from the third pouch, coursing dorsal to the carotid axis and then extending lateral to these vessels to the pericardium. In older diagrammatic representations (see Fig. 9) a different course is shown for a third pouch branchiogenic fistula (which is shown taking the expected course) and the thymic duct. The thymic duct is shown emerging from the thyrohyoid membrane, consistent with third pouch origin, but it then turns down medial to the carotid axis. Wenglowski admittedly could not follow the complicated development of the carotid vessels and discounted them as guides as to origin of fistulae. He described the thymic duct as coursing "contiguous" to the vascular sheath, but does not specify as to whether it is contiguous dorsally, medially, laterally or ventrally. It seems logical and consistent, however, in the light of Congdon's (1922) description of the aortic arch system and Frazer's description of the course of the thymic duct, to hold to these criteria in interpreting the origin of fistulae.

The thymus briefly consists of a cortex and medulla, not sharply differentiated, and contains lymphoid tissue, Hassall's corpuscles and a stroma. The origin of these elements, entodermal or otherwise, is controversial. Even the syncitial portion of the connective tissue and lymphoid cells have ad-

herents claiming for them entodermal origin.[9] Muscle and glandular tissue are not a part of normal post-embryonic thymus tissue, though commonly found in lateral fistulae.

The origin of Hassall's corpuscles, the distinctive constituent of thymic tissue, is most controversial. Frazer points out that there develops in some lower mammals a "superficial thymus" in association with the ectoderm of the third cleft. His, in 1881, thought the thymus developed from the floor of the cervical sinus in the region of the third cleft, but later changed his mind. Wenglowski in 1913, in attempting to explain stratified squamous epithelium in complete lateral fistulae which he claimed were vestiges of the thymic duct, stated the thymic tract is in relation to the third cleft in the sinus and has been seen to join with this ectoderm, and mentions this ectoderm as a possible source of Hassall's corpuscles. He stated, however, that this taking on of ectoderm from the cervical sinus occurs only in those cases of fistula in which ectodermal, as well as entodermal, epithelium lines the tract.

Norris[10] in 1938 described what he terms the discovery of the origin of Hassall's corpuscles. He states that the ventral horn, or angle of the third pouch, contacts the ectoderm of the third branchial cleft in the precervical sinus, and that a portion of this cleft buds and joins the thymic anlage as an onlay covering the entodermal thymus cord to form the cortex containing the Hassall's corpuscles. Maximow and Bloom,[11] in their textbook of histology, state however, that Hassall's corpuscles are predominantly medullary. Garrett[12] in 1948 was unable to corroborate Norris' claim of a contribution of sinus ectoderm to the thymus; in fact, Garrett found that the thymic anlage coursed near, but received no contribution from the placodal duct of the Xth nerve rather than the third cleft, and took exception to Norris' illustrations on this point. It is evident that an ectodermal contribution to the thymus has not been unequivocally established.

Aside from the rare possibility of a persistent thymic duct, it is not unusual for bits of thymic tissue to remain in the cervical region, posterior and lateral to the thyroid lobes, or associated with parathyroid III (Ungerson, 1906; Gerard,

1928; Norris, 1937). Conversely, a case with parathyroid tissue within the thymus is on record (Churchill and Cope, 1934). Failure of descent or a true cervical thymus, on the other hand, is rare.

In summary, what should distinguish a complete fistula of third pouch origin from a fistula of thymic duct origin? For both, they should open internally in the pyriform fossa, pierce the thyrohyoid membrane and course dorsal or caudal to the carotid axis. Frazer traces the course of the thymic duct as lateral to the great vessels to the thorax, while Wenglowski traces the course as "contiguous" to the vascular sheath and then along the anterior border of the sternomastoid. A third pouch fistula, or a second pouch fistula for that matter, has an external opening embryologically through the corresponding branchial groove. A thymic duct normally has no external opening, and Wenglowski, as will be discussed under "The Refutation of the Thymic Duct Theory," explains this as due to secondary suppuration.

The fourth pouch contributes parathyroid IV. A vestige of fourth pouch origin, because of its relations to the aorta of the fourth branchial arch, would have to loop around the aorta on the left or the subclavian artery on the right, as these are fourth arch aortae derivatives (see Fig. 6). No such relationship has ever been reported for a fistula; also, should such a fistula exist, it would course caudal to the superior laryngeal nerve, the nerve associated with the fourth arch.

THE DISAPPEARANCE OF THE PRECERVICAL SINUS.

Initially the four external arches are equally distinct on the ventral aspect of the head. The first and second arches grow more rapidly, and soon the precervical sinus is a depression between the second arch cephalad and the caudal margin of the sinus field, the precursor of the posterior neck and chest wall (see Fig. 1-A). In this depressed area are the third and fourth arches and the grooves of the second, third and fourth arches. His, in 1881, described the formation of the precervical sinus as a telescoping process, cephalad to caudad, with the second arch overriding the third, and the third the

fourth, until in this manner the second groove and the more caudal arches and grooves are covered in by the second arch. Rabl, in 1886, claimed the third arch did not override the fourth. Rabl, in 1907, described a fusion between the second arch and the thoracic wall precursor as the means by which the sinus is closed. According to Frazer, Rabl worked with moles and not human embryos.[4]

The following description of the closure of the precervical sinus, based on Rabl's work, has for a long time been accepted. The sinus is at first widely open laterally. As the second arch grows caudally the opening of the sinus is narrowed to a duct, the precervical duct. After closure of the duct externally, a closed space is formed, enclosing the second, third and fourth grooves, and the third and fourth arches (see Figs. 6, 10). If one of the closing membranes breaks through, a communication is established between the primitive pharynx and the sinus, and if the precervical duct remains patent, a complete fistula is established. With further growth of the embryo, the grooves entering the sinus are elongated into ducts and the arrangement described is that of the third and fourth ducts joining to form the precervical duct with the second groove duct opening into the neck of this precervical duct (see Fig. 6). Eventually the ducts and sinus are narrowed and obliterated. The explanation of whether a complete fistula opens high or low in the neck, based on this concept, depends on the length of the precervical duct; however, Wenglowski, Frazer and Garrett maintain, as shown below, that the third arch is not obliterated in the precervical sinus, and so makes difficult an explanation of the external opening of lateral fistulae based on a breakthrough of the second closing membrane communicating externally via the duct of the precervical sinus. Wenglowski and Frazer hold that while the second arch grows much larger than the third, the third is not telescoped by it, and it is the third arch which fuses with the thoracic wall. Wenglowski's contribution was that he first noted that the third arch was not obliterated in the precervical sinus development. He erred, however, in maintaining that the third arch could not be represented postembryonically below the level of the hyoid. Actually, the hyoid

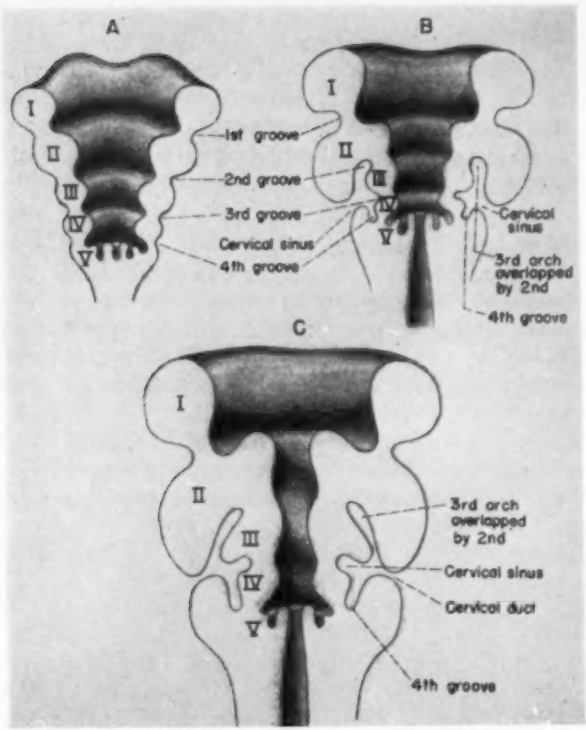

Fig. 10. Showing early development of the pharyngeal wall; A, Showing proximity of ectodermal floor of branchial clefts to entodermal pharyngeal pouches; B. Rapid growth of the (hyoid) arch II overshadows postoral arches forming the cervical sinus; C. Occlusion of the cervical sinus.

Note: This is based on the old concept of Rabl. Contrast with Figs. 11, 13, 15.—(As redrawn by Proctor[21] from Ward and Hendricks[18]).

is formed from both the second and third arch cartilages, with the second arch forming the lesser cornu and the third arch the greater cornu and body; thus a second pouch origin fistula passes above the hyoid but posterior to the lesser cornu. The level of the second or hyoid arch is more accurately described as that of the posterior belly of the digastric and stylohyoid muscles, which are second arch derivatives, innervated by the VIIth nerve. The course of lateral fistulae over

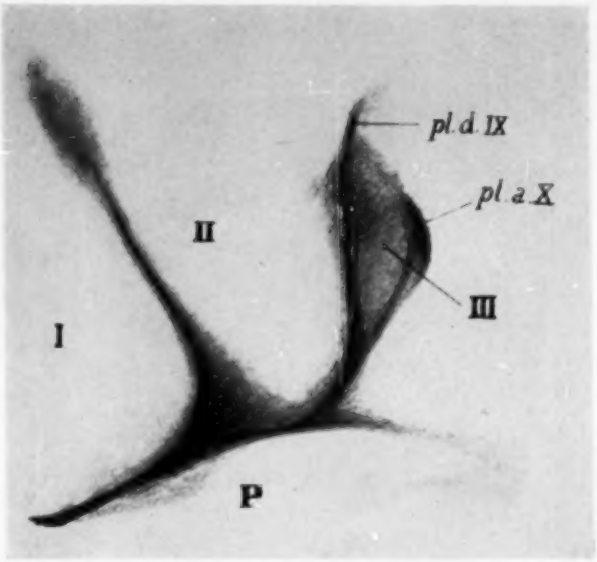

Fig. 11. Region of the precervical sinus in a 10 mm. embryo; left side. I, II, III, branchial arches; P, pericardium; pl. d. IX, placodal duct of IXth nerve; pl. a. X, placodal area of the Xth nerve. The widely open placodal recess of the vagus, pl. a. X, is below and behind the narrow duct, pl. d. IX, leading to the glossopharyngeal ganglion. The epipericardial ridge is very prominent below and behind the third arch. The shaded area at the upper end of the first groove will develop into the pinna; the second groove will become the transverse flexure line of the neck, and the ectoderm over arch III will occupy the approximate region of the anterior triangle of the neck. Visualize, as the head lifts from the pericardium, the region I, II and III rotating clock-wise with the fulcrum at III (see Fig. 16).—(From Frazer[4]).

the hyoid, dorsal to the lesser cornu and beneath the posterior belly of the digastric, was ignored by Wenglowski in his claim that these were more properly of thymic duct (third pouch) origin.

Frazer,[4] in 1926, working with human embryos, confirmed that the third arch is not obliterated in the precervical sinus, and in addition showed by logical inference that it is represented on the post-embryonal neck as the approximate region of the carotid triangles (see Fig. 5). Dorsally the sinus area is bounded by the paraxial tissues delimiting the upper extremities of the third and fourth arches and their respective

grooves, which make up the depressed area of the cervical sinus. This paraxial tissue is continued caudally and ventrally. The ventral continuation of this boundary tissue is known as the epipericardial ridge (see Fig. 11) and is interposed between the ventral extremities of the second, third and fourth arches and the pericardium. The ridge passes medially

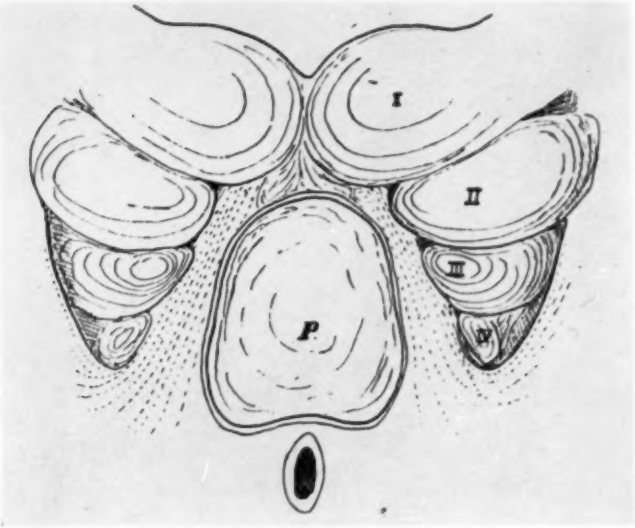

Fig. 12. Semi-schematic view of the lower aspect of the pharyngeal region, the pericardium having been removed, save for its roof. I, II, III, IV, pharyngeal (branchial) arches; P, roof of pericardium. The whole region is somewhat flattened out to show the relations. Between the pericardium and ventral arch extremities is shown the extension of the epipericardial ridge (dotted lines). In this area will develop the tongue muscles and hyoid rudiment.—(From Frazer[4]).

beneath the ventral extremity of the third arch, but the tract of cells forming the ridge continues forward to pass internally to the lower end of the second arch. The ridge thus connects the arches with the pericardium as far forward as the level of the second arch. In the ridge are found structures vital to this discussion, namely the sternomastoid and infrahyoid muscles and the XIIth nerve. Within its area is also de-

veloped the clavicle, and ventrally its cell tracts contact the hyoid bone rudiment.

Only the first or mandibular arches meet ventrally in the midline. The ventral extremities of the more caudal arches are separated, with the gap progressively wider for the more caudal arches. In apposition to this area of gapping is the pericardium (See Fig. 12). The epipericardial ridge, as already described, turns medially beneath the ventral extremities of the third and second arches and then its cell tracts course between the pericardium ventrally and the pharynx dorsally to bury themselves in the mandibular arches. In this interval between the arches ventrally are developed the tongue muscles and hyoid bone rudiment.

At this early stage, Frazer calls attention to the presence of areas of thickened ectoderm, the placodal areas, in the paraxial boundary tissue dorsal and caudal to the sinus field. This ectodermal thickening later becomes anatomically related to the nerve rudiments of the deeper lying ganglia of the IXth and Xth nerves. Similar more cephalad thickenings are related to the Vth and VIIth nerve ganglia. They appear to make no vital contributions to the nerves mentioned, and Frazer admits ignorance of their possible function. Arey[13] states that epibranchial placodes originate at the dorsal ends of the branchial clefts, and in some lower vertebrates cellular proliferation from them clearly adds to the neighboring ganglia. In higher animals and man, Arey states that the relations are less plain, and indications of contributions to the ganglia is in dispute. Regardless, these ectodermal placodes overlay the upper part of the third arch and adjoining second cleft in relation to the deeper lying IXth nerve ganglion and encompasses also the greater part of the fourth arch and the caudal boundary of the field in relation to the Xth nerve ganglion. By a process of thickening and infolding the placodes are first recessed and then form ducts leading to expanded vesicles in relation to the respective nerve ganglia. Eventually the vesicles and ducts are obliterated. In this process of thickening and infolding the structures of the cervical sinus are secondarily and passively affected. The fourth arch is completely engulfed. The duct of the IXth placode

can be seen (see Fig. 11) to be located above the third arch in relation to the dorsal extent of the second groove. According to Garrett, it forms an appendage to the second cleft. Frazer states the thickening and infolding bulges above but does not cover or invade the third arch area. Garrett,[12] in confirming and enlarging on Frazer's findings in 1948, states they have since been completely overlooked in the anatomical literature, and almost so in the clinical literature. What older writers described as the precervical sinus disappearing through a fusion of the second arch with the thoracic wall, with the sinus reduced to an ectodermally lined cyst opening on the surface by a duct, has been disproven. The precervical sinus itself is never closed off to form an ectodermal lined cyst; only the placodal ducts are involved. One of Garrett's microphotographs in particular startlingly resembles the description of the precervical sinus duct arrangement of earlier writers; namely, that of the third and fourth clefts opening into the duct of the precervical sinus with the second cleft opening into this duct. What appears to be the fourth cleft, however, is labeled the placodal duct of the Xth nerve with the third cleft obliterated, but with the third pouch appended to it but separated by a thin layer (see Fig. 13).

The disappearance of the third arch as a projection on the surface is explained on the basis that the arch is concerned in the formation of many important structures in and about the pharynx, but not on the surface; and its appearance in the first place is only the result of its growth and condensation when the wall of the pharynx is relatively thin. The second groove, on the other hand, outlines and maintains the prominence of the second arch because it becomes the transverse flexure line of the neck. The second arch remains prominent posteriorly also, because from it and the adjoining first arch posteriorly are developed the pinna (see Fig. 14).

To summarize Frazer's contributions here, he demonstrated, first: that the cervical sinus was not obliterated by a fusion of the second arch with the thoracic wall precursor to enclose an ectodermal sac. Rather, he showed that the branchial structures involved in the sinus are passively affected in the development and obliteration of the placodal ducts of the IXth

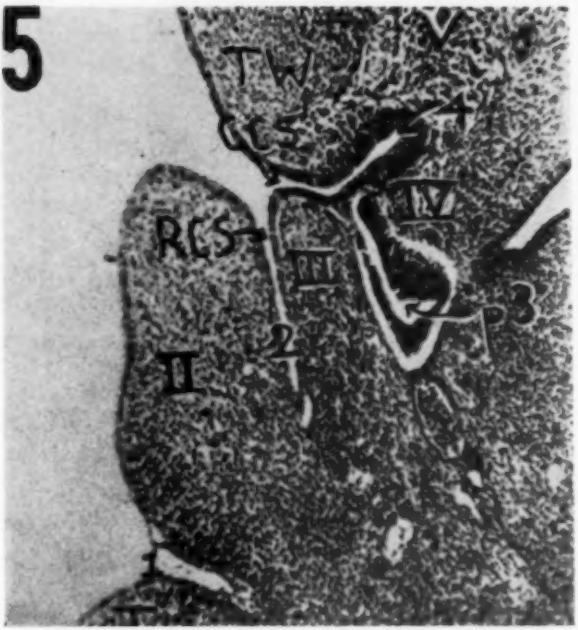

Fig. 13. Human embryo, 10.5 mm., right side, x 52. Third cleft obliterated. I, II, III, IV, branchial arches; 1, 2, first and second branchial clefts; 4, fourth branchial cleft engulfed by placodal duct of Xth nerve; p-3, tip of third pharyngeal pouch; RCS and CCS, rostral and caudal parts of the cervical sinus; TW, thoracic body wall; V, vagus nerve. In the preceding microphotograph in Garrett's paper, that of an 8 mm. embryo, the placodal duct of the IXth nerve is shown as a dorso-caudal evagination from the second cleft into the tissue of the third arch to come into relation with the glossopharyngeal trunk. The placodal vesicle of the Xth nerve reaches its greatest prominence later, at about 12 mm., when the last traces of its connection with the cervical sinus become obliterated.—(From Garrett, F. D.: Development of the Cervical Vesicles in Man.[12] Fig. 1, Plate I, Anatomical Record, 100:111.)

and Xth nerves, or expressed simply, the cervical sinus obliterates by a fusion of its ectodermal lining from within toward the surface. Second, he demonstrated that the third arch contributes to the formation of the ventral portion of the neck; namely, the area of the carotid triangles. In effect, nevertheless, the third arch fuses with the precursor of the posterior neck and thoracic wall.

At the time of the last flattening out of the third arch the

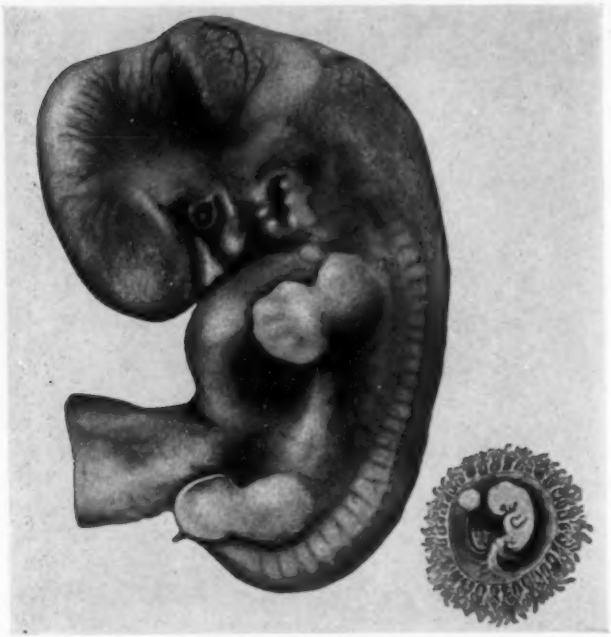

Fig. 14. A. Human embryo a little over six weeks (14 mm.); retouched photo. The pinna is shown forming about the first groove posteriorly; the head still remains curled over the pericardium, the neck has not formed and the mandible has not grown sufficiently to force the pinna dorsally. (From Patten,[9] by courtesy of the Carnegie Institution of Washington.)

sternomastoid is distinct in the caudal border of the sinus field (see Fig. 15); also, coursing in this caudal boundary, but at a deeper level, and then turning to course ventrally to the sinus field in the epipericardial ridge, are the tongue muscles and the accompanying XIIth nerve. It follows from this that any vestigial structure from the arch area must course ventral to the XIIth nerve; also, as the head grows away from the pericardium as the neck develops, and the XIIth nerve travels dorsally in the outer layer of the mesoderm of the body wall to its final position, it would engage any structures coursing laterally. Frazer states that the sternomastoid branch of the

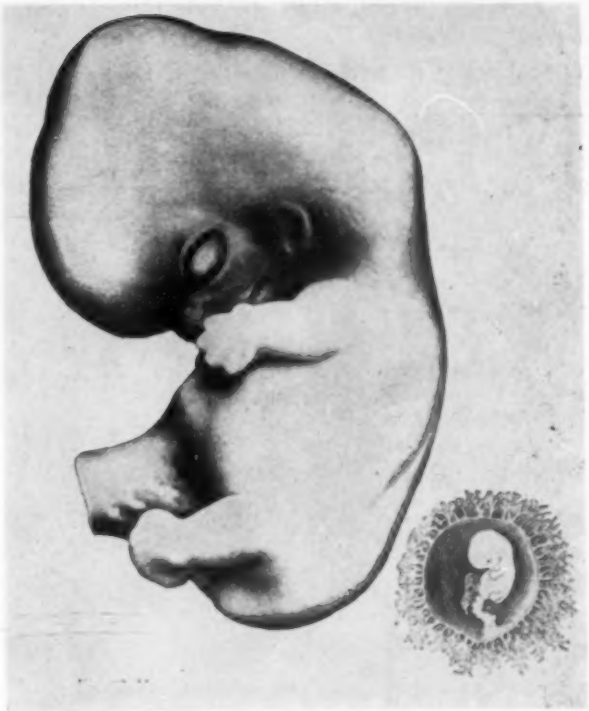

Fig. 14. B. Human embryo of about seven weeks (17 mm.). The head is lifting from the pericardium as the neck is forming, the mandible is evident, and the pinna is moving toward its final location. (By courtesy of the Carnegie Institution of Washington.)

occipital artery coursing laterally in the path of the XIIth nerve is engaged by it, and so the XIIth nerve comes to hook around this vessel in the neck (see Fig. 5). Sometimes the parent vessel, the occipital artery, is hooked around. It also stands to reason that a second pouch and cleft vestige would course ventral to the IXth and Xth nerves. A third pouch vestige would emerge caudal to the IXth nerve and ventral to the Xth nerve.

THE NECK FORMATION AND A THEORY EXPLAINING THE EXTERNAL OPENING OF FISTULAE NEAR THE STERNOCLAVICULAR JOINT.

As noted above, the neck becomes evident as ventral growth becomes more active and separation from the pericardium is effected. As this ventral growth starts, there are important changes rapidly taking place beneath the ventral extremities of the second and third arches in the epipericardial ridge and

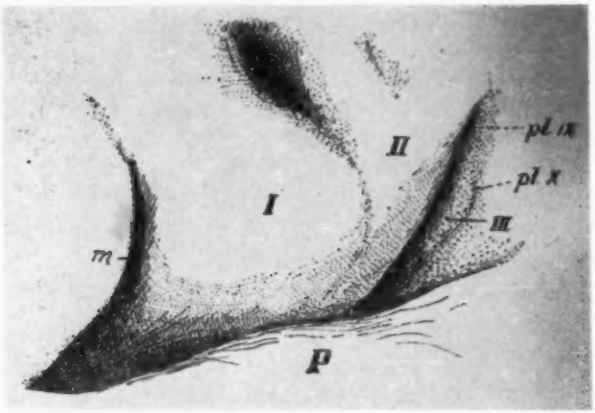

Fig. 15. Left side of region in an embryo of 12 mm., showing the structures behind the mouth, m. I, II, III, branchial arches; P, pericardium; ectodermal connections with placodal cysts indicated at pl. IX, pl. X. The third arch is less prominent, and is fading into the caudal boundary of the sinus field and epipericardial ridge with their contained sternomastoid muscle, which serves to bound the anterior triangle of the neck posteriorly and inferiorly.—(From Frazer[4]).

its environs. The hyoid rudiment forms in the space between the ventral ends of the second and third arches. The clavicular rudiment forms close by just caudal to the second groove (precursor to the transverse flexure line of the neck), where it is continuous with the episternal thickening ventral to the upper pericardium. The infrahyoid muscles are in the epipericardial ridge here. The sternomastoid muscle extends along the continuation of the caudal margin of the sinus field into the epipericardial ridge to contact the clavicular rudi-

ment condensation. It is evident that the second groove at its ventral extremity is here in intimate relation with the clavicle and the infrahyoid and sternomastoid muscles. The relationship is most pertinent (see Figs. 15, 16). As ventral growth proceeds the infrahyoid muscles, fixed to the hyoid above and the clavicle below are drawn out on the ventral

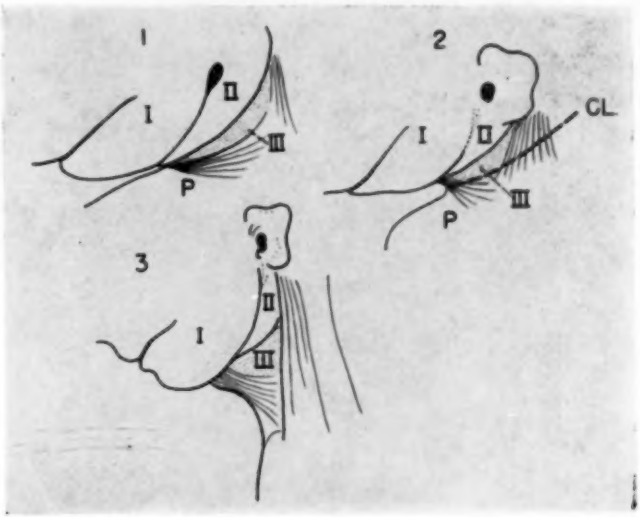

Fig. 16. Three schemes to show the situation acquired by the third arch as the neck forms. Cl. gives the general position and line of the clavicle; I, II, III, branchial arches; P, pericardium.

Note: Visualize a persistent opening at the ventral end of the second groove in Scheme 1; then consider this opening trapped or maintaining its relation with the thoracic wall near the letter P. As the infrahyoid muscles are drawn upward as the hyoid moves upward, this opening is brought into relation with the anterior border of the sternomastoid; the infrahyoid muscles rotate from a horizontal to a vertical position and the sternomastoid is drawn ventrally at its clavicular attachment.—(From Frazer[4]).

side. The sternomastoid bounding the third arch caudally is a fixed structure behind. The flexure line of the neck marks off the second and first arches cephalad, so that the third arch area by logical inference practically corresponds with the skin covering the carotid triangles. Frazer does not contend that this correspondence to the carotid triangles is

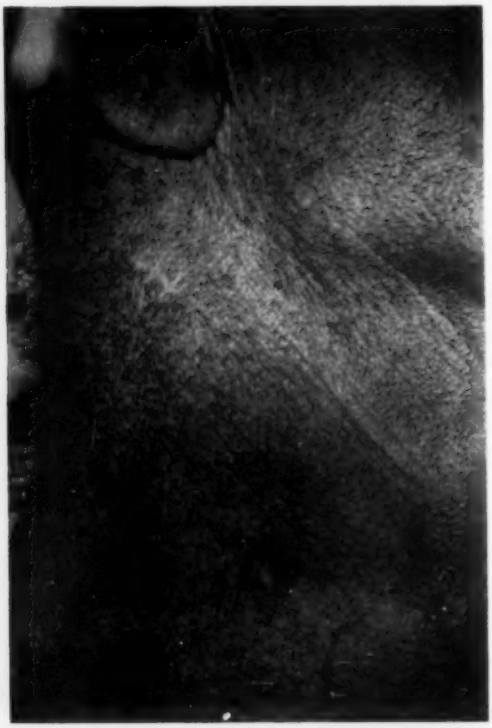

Fig. 17. Photograph of a cervical cartilaginous bar. Note that the bar is not at the anterior margin of the sternomastoid. In this connection attention is called to Frazer's remark that the sternomastoid marks the posterior margin of the sinus field and that the third arch represented ectodermally on the neck is in the approximate region of the anterior triangle. Also see under discussion of atypical external openings in: Bailey, H.: The Clinical Aspects of Branchial Fistulae, British Journal of Surgery, 21:173, 1933.

an exact one. An external fistulous opening related to the posterior margin of the sternomastoid has been reported, in fact.[22]

A cartilaginous bar in the general region of the carotid triangles, variously termed a cervical or auricular appendage, may well be a remnant of the cartilage of the third branchial arch. Such a case is illustrated (see Fig. 17). The pinna

is developed above and below the posterior extremity of the first (hyomandibular) branchial cleft from the first and second branchial arches respectively. In Fig. 14 it can be seen that the pinna when in its formative stages, before the neck is developed, is relatively ventral in location and close to the thoracic wall. Arey[13] states that it persists in this location rarely, and when it does is in combination with agenesis of the mandible, with the mandible failing to force the ears laterally to the sides of the head. It would seem, therefore, that with Frazer's demonstration of the persistence of the third arch on the neck, cartilaginous bars in this region would be more likely explained as due to a vestige of the third arch rather than a remnant of the pinna left behind. Hyndman and Light[5] and Brown,[14] state that small bits of cartilage are often found at the external orifice of lateral fistulae. These may be of second or third arch origin, and strengthen the case for branchial as against thymic duct origin.

It can be seen from the above that branchial remnants can exist below the level of the hyoid. A breakthrough in the third groove, as well as one from the second, could be related to the infrahyoid and sternomastoid muscles in the approximate region of the carotid triangles. A fistula of third membrane breakthrough is primarily less likely from an embryological standpoint (see section on closing membranes); second, it would have to course posterior to the common-internal carotid axis and open internally in the pyriform fossa. A breakthrough of the second closing membrane is more likely embryologically, and if it emerges in the ventral extremity of the second groove is closely related to the clavicle, infrahyoid and sternomastoid muscles (See Figs. 15, 16). Its proximal relations would have to be either between the internal and external carotids or anterior to the carotids, and it would have to pass beneath the posterior belly of the digastric muscle, and open internally in the region from and including the tonsil fossa to the Eustachian orifice. Perusal of the clinical literature indicates this latter set of relations holds for the usual lateral fistulae.

Why is it that branchial cysts are found only in the upper

third of the neck, while lateral cervical fistulae can open almost to the clavicle? I have presented a theory of the development of fistulae from a breakthrough of the second closing membrane at the time and place when the second cleft and the rudiments of the clavicle, infrahyoid and sternomastoid muscles are grouped close together. It must be assumed that when an external opening occurs into the ectoderm at the base of the future neck, this opening is trapped in an area of relatively slow ectodermal expansion. As the cephalic attachment in the region of the tonsil fossa grows away from this area in the ventral expansion, causing the formation of the neck, the fistulous tract is drawn out as are the infrahyoid muscles. Where no caudal anchorage occurs, as in a branchial cyst or incomplete internal fistula, the vestige is carried upward or cephalad, maintaining its relations at the approximate hyoid level.

REFUTATION OF THE THYMIC DUCT THEORY.

Wenglowski[3] presented his thymic duct theory of the origin of lateral fistulae in 1913. Wenglowski stated that there was such a uniformity in lateral cervical fistulae that a common origin must exist. He concluded, however, from his studies of human embryos that no vestige of the branchial arch apparatus could exist post-embryonally below the level of the inferior border of the hyoid bone. This was based on his contention that the initial dorso-ventral relationship of the arches persisted; namely, the third and fourth arches remain dorsal to the second arch, and so no arch field derivative could persist caudally below the level of the second arch. This has never been corroborated, and as has been shown by Frazer,[4] the third arch is represented on the neck in the region of the carotid triangles. For this same reason, Wenglowski reasoned that any fistula of branchial origin could not turn down to reach a level below the level of the hyoid, but should course almost straight laterally to open in the region of the angle of the jaw. Because most fistulae, however, do angle to course inferiorly and open on the neck, sometimes at the sterno-clavicular region, Wenglowski sought to find another explanation for fistulae, and came up with the thymic

duct theory, as the thymic duct normally leaves the branchial area to course inferiorly.

How does Wenglowski explain the external opening of a fistula on the basis of thymic duct origin? A branchial fistula through the groove opens externally in the embryo, and if this condition persists there is a ready-made explanation for an external opening. Wenglowski states that fistulae open externally only by secondary suppuration. He reasons that only a thymic duct vestige would consistently open along the anterior border of the sternomastoid as this is related to its normal course, while a branchial fistula could just as easily open posteriorly in the neck, depending upon which haphazard direction the pus burrowed before pointing externally. While it is true that many fistulae show evidence of inflammation, I know of no other author who expressedly feels that this is the usual modus operandi of external opening of congenital fistulae.

How does Wenglowski get around the fact that the internal opening of lateral fistulae is in the second pouch area, let alone the course above the hyoid, which he accepts as of second arch origin in part? He concedes that the tonsil fossa proper is of second pouch origin. He argues, however, that fistulae opening in this region open not into the fossa proper but in relation to the palatoglossus muscle of the anterior pillar, which he claims is of third arch origin, innervated by the IXth nerve. The cases of Dowd,[15] Kleinert,[16] Blassingame,[17] and the author, however, exhibited openings within the tonsil fossa proper. Morris' Textbook of Anatomy states the palatoglossus muscle is innervated by the Xth nerve, the nerve of the fourth arch, while Gray's textbook states it is innervated by the XIth nerve; no mention is made of the IXth nerve.

Wenglowski decried Kanstanecki and Milecki,[3] who emphasized nerve and blood vessel relations as more significant than muscle relations. He stated that the development of vessels in this region was so confusing that no useful relation could be established. Frazer and Ward and Hendricks,[18] however, state that the relations to nerves and vessels are fixed, and

barring vessel anomalies themselves, are the only constant guides as to origin of vestigial structures. Wenglowski's paper erroneously states that the artery of the third arch is the external carotid.

In further discussion Wenglowski argues that if a fistula is of branchial origin it should be lined by entodermal or columnar epithelium in its inner portion to correspond to the pharyngeal pouch portion, and by ectodermal or stratified squamous epithelium in its outer portion to correspond to the branchial groove, and that this arrangement does not occur; however, there are cases with just this arrangement, though they are not the rule. Rudberg,[19] *e.g.*, cites several cases lined with stratified squamous epithelium which changed to ciliated columnar near the pharynx. In fact, Rudberg found that squamous epithelium occurred in 90 per cent of cysts and fistulae, ciliated columnar, especially near the pharynx, in 8 per cent, and both types of epithelium in 2 per cent. Carp and Stout[6] report branchial cysts with both types of epithelium side by side. Wenglowski then found himself in the position of having to explain stratified squamous epithelium, supposedly indicating ectodermal origin, in thymic fistula and as noted in the section of "Fate of the Pharyngeal Pouches," explained it as based on contact with the cervical sinus ectoderm only in those cases where "ectodermal" epithelium occurred. Actually, however, Maximow and Bloom[11] state that experimental studies of vertebrates show that many of the cells in the embryonic germ layers have no inherent specificity during early developmental stages, and that if they are moved from one place to another they will often develop in conformity with their new surroundings.

Wenglowski has this to say about the presence of Hassall's corpuscles as evidence of thymic origin, and I translate as literally as possible: "In cysts of the lower half of the neck are found clumps of flat epithelium which are similar to the Hassallian corpuscles." There is no specific case citation, nor is there reference to their ever having been found a complete fistula rather than a cystic structure, nor is there any statement as to the incidence of finding Hassall's corpuscles. Much emphasis is placed on the wealth of lymphoid tissue

which he states shows close relationship to the thymus. What does this imply as regards the usual lymphoid tissue in cysts above the hyoid level, which he will not contest as being branchial in origin? Lymphoid tissue is commonly associated with the pharynx.

The author has encountered no clinical description of a complete fistula meeting the accepted criteria of third pharyngeal pouch origin, either thymogenic or branchiogenic. This is confirmed by Dowd[15] quoting McKenty, Whitacre and Konig, and also by Taylor and Burwell.[8] Rudberg reports a case in a 13-year-old girl presenting a swelling in the neck between the larynx and the carotid sheath which was found to lie in front of the sternomastoid with a small deeper ramification extending behind the common carotid, where it divided into ascending and descending branches. The ascending branch ended blindly at the level of the carotid bifurcation, and the descending branch narrowed to a string ending in a terminal enlargement about a finger-breadth above the clavicle. On microscopic examination the mass consisted of a few irregular cystic cavities lined with both flattened and stratified squamous epithelium, with considerable lymphoid tissue beside the cyst walls, and in this lymphoid tissue were a large number of small, rounded bodies resembling Hassall's corpuscles. Rudberg stated most surgeons would consider this thymogenic, but, from its vessel relationship he considered it as a third branchial groove cyst with the additional presence of thymic tissue. In the author's opinion, due to the absence of an external opening, this could well be a thymic duct vestige. Certainly, the vascular relationship indicates third pouch origin, whether branchiogenic or thymogenic. This case, however, is the exception that "proves the rule." Wenglowski developed his theory in explanation of the usual type of fistula, which as has been repeatedly demonstrated, opens in the region of the tonsil fossa, passes over the hyoid dorsal to the lesser cornu and passes either between the carotids or ventral to the carotid axis and opens externally near the sternoclavicular junction. Rudberg's case meets the criteria of a third pouch-derived cyst and to prove it, exhibits the proper vessel relationship and demonstrates Hassall's cor-

puscles. If it exhibited Hassall's corpuscles and had vessel relations associated with accepted criteria of second pouch origin, then Wenglowski's theory would have some justification and embryologists would have to start over again.

HISTORICAL SUMMARY.[20,3,5,6]

Huncyowski, in 1789, probably reported the first cases of lateral cervical fistula. The first accurate description of the branchial apparatus was in vertebrates by Rathke in 1825. He noted the resemblance of the branchial clefts to the gill apparatus of sharks but discarded any association phylogenetically. Ascherson, in 1832, distinguished between median and lateral fistulae, and first associated lateral cervical fistulae with incomplete closure of the branchial clefts. Cysts were included as branchial derivatives by Roser in 1859. Heusinger, in 1865 and 1876, reported that all fistulae opened internally near the base of the tongue and opened at various levels of the neck externally depending upon which branchial groove failed to close. Cusset (1877) and Bland-Sutton (1895) presented different schema but based on identification of groove origin by level of external opening also.

The next major contribution was by His, in 1881. He used human embryos and first used the more precise reconstruction method of studying them. He described the closing membranes and noted their failure to break through normally, as in aquatic animals to form gill slits. He described the precervical sinus and the triangular area ventral to the pharynx between the ends of the arches occupied by the pericardium; the mesobranchial field of His. The prefix "pre-" in the term precervical sinus was intended to refer to a ventral orientation with regard to the neck (Rabl accused him of implying an oral orientation).

Rabl, in 1886 and 1907, described the closing membrane of the second pouch and groove as two layers of epithelium in contact and mentioned the proclivity for a breakthrough here to form a fistula. He discounted a third membrane breakthrough possibility as a relation to the thymus or carotid body would have to exist. A fourth membrane breakthrough was

felt not possible because of the thick layer of mesoderm intervening. To him is ascribed the concept of the disappearance of the precervical sinus by fusion of the second arch with the thoracic wall, and the formation of the precervical duct as an explanation of the external opening of lateral fistulae. Konstanecki and Milecki, in 1890, organized the literature to their time and were instrumental in discarding the schemes of Heusinger, Cusset and Bland-Sutton as not corresponding to anatomical facts. They established the concept that the inner opening determines the origin of fistulae which is branchial, with the opening always in the inferior portion of the tonsillar fossa at the root of the tongue and so of second pouch origin.

Wenglowski, in 1913, presented his thymic duct theory of the origin of lateral cervical fistulae with his major premise that no branchial vestige could remain beneath the level of the inferior margin of the hyoid bone. His thymic duct theory has several weaknesses, as described. His worthy contributions appear to be that he confirmed Hammar's (1902) findings that the points of contact of groove and pouch in the various closing membranes are not extensive and vary with the several membranes, even between sides of the same embryo, but that the most constant closing membrane, other than the first, is that of the second. He also first described how the third arch was not obliterated in the precervical sinus.

Frazer, in 1923 and 1926, demonstrated that the third arch not only was not obliterated in the precervical sinus, but also took part in the formation of the neck corresponding with the carotid triangles. He also stated that the precervical sinus was not obliterated by a fusion of the second arch (Rabl) with the thoracic wall, but that the structures involved in the sinus are passively affected in the development and obliteration of the placodal ducts of the IXth and Xth cranial nerves. He also demonstrated that all branchial vestiges must develop ventral or anterior to the XIIth nerve. He also described the area from and including the tonsil fossa to the Eustachian orifice on the lateral pharyngeal wall as the postembryonal derivative of the ventral angle of the second lateral

pharyngeal pouch. He would not conjecture as to the origin of vestiges in this region except in broad terms. He mentioned the possibilities of the placodal ducts, as well as the grooves, under the heading of "abnormal persistence of normal states" or "delayed development" as well as "abnormal processes of development." Fistulae opening near the clavicle, he feels, could possibly derive by either method. He apologizes for having ruined a duct system as part of the precervical sinus as a source of explanation.

CASE REPORT.

Mr. J. B. W., white, age 33, was admitted Dec. 22, 1951, with a diagnosis of bleeding duodenal ulcer. On routine physical examination the examining physician noted a small sinus above the sternal attachment of the sternomastoid which seemed to be attached to a cord under the skin, which ran cephalad. On milking the cord the physician reported a clear mucus exuding from the sinus opening. The patient was referred to the E.N.T. clinic. There was a history of drainage from the sinus opening as long as the patient could remember, and the secretion was usually clear. The patient does not recall ever having a sore throat. A probe was passed to the upper level of the thyroid cartilage and passed beneath the sternomastoid at about its midpoint. Probing caused coug'hing, and a foreign body sensation in the throat at about the level of the superior border of the thyroid cartilage. Lipiodol was instilled and X-rays made. These revealed a fistulous tract with no cystic enlargements in its pathway. The tract passed over the hyoid just posterior to the lesser horn and then turned medially toward the pharynx. On injecting the fistula with methylene blue, dye was seen to come from a large crypt in the upper pole of the right tonsil. The tonsils were normal in appearance. The fistulous tract was excised without difficulty via two step-ladder incisions. The right tonsil was removed in the usual manner and the tonsil bed sutured. There was no contact with the great vessels, and the tract became quite thin as it approached the tonsil fossa. Dissection was simple with no evidence of inflammatory reaction. Postoperative course was uneventful and a questionnaire three years later revealed the patient was asymptomatic as regards this lesion.

The gross description was that of a tubular structure measuring 10.5 cm. in length and varying in diameter from 4 to 6 mm. An elliptical fragment of skin was noted attached to the specimen. Microscopic description was as follows: The tract is lined by stratified epithelium which does not show keratinization. The epithelium is supported by connective tissue. Definite smooth muscle structures are not seen. There is very slight lymphocytic infiltration in this supporting connective tissue. No structures similar to Hassall's corpuscles are seen. Occasional fragments of salivary gland tissue are seen at various levels adjacent to the connective tissue. This structure is surrounded by voluntary muscle and connective tissue. Microscopic diagnosis was, "fistulous tract."

SUMMARY.

1. All evidence points to the second closing membrane breakthrough as the most likely source of lateral cervical

vestigeal fistulae and cysts below the mouth and ear regions. Cysts and fistulae are of similar origin.

2. The ventral angle of the second pouch is represented in the adult lateral pharynx as the region from and including the tonsil fossa to the Eustachian orifice. A congenital fistula, with an internal opening anywhere in this region can be assumed to be of second pouch origin if it complies with nerve and vascular relationships. If ever encountered, a third pouch fistula would open internally in the pyriform fossa and a fourth pouch fistula would open still lower, beneath the fold of the superior laryngeal nerve.

3. A second pouch fistula courses over the hyoid, dorsal to the lesser cornu. It then commonly passes between the internal and external carotid arteries, but may pass anterior or ventral to both. An anterior course may be determined by preoperative X-ray of the tract filled with radio-opaque material. An anterior course predicts a relatively simple operative procedure. Embryological correlation of these variations is discussed.

4. The relation to nerves and vessels is the determining factor in identifying the origin of a vestige, as well as the site of internal opening. All branchial vestiges are found ventral to the XIIth nerve. Second pouch fistulae should course ventral to the IXth and Xth nerves also.

5. A third pouch vestigeal structure would have to open internally in the pyriform fossa, course beneath the hyoid and pass dorsal to the internal or common carotid artery. No such relationship for a *complete* fistula has ever been reported. A fourth pouch fistula would have to loop around the aorta on the left or the subclavian artery on the right, and again no such relationship has ever been reported.

6. The thymic duct theory of Wenglowski as an explanation of the usual fistula under discussion is not in keeping with clinico-pathological and embryological facts.

7. Embryologists list many possible sources of vestigeal structures,[21] and while they suggest the capability of producing vestigeal structures, only the second pouch and cleft

vestiges are significant clinically, with the exception of cartilaginous bars (see below).

8. The older concepts of the disappearance of the precervical sinus and the development of the neck appears to have been displaced by Frazer's work with human embryos. The precervical sinus is passively affected in the development and obliteration of the adjacent placodal ducts (IX and X), with the sinus obliterating by a fusion from within toward the surface. At no time is it represented by an ectodermal lined sac; all such simulation, according to Frazer and as confirmed by Garrett, is due to confusion with the ectodermal lined placodal ducts.

9. The third arch is represented on the post-embryonic neck as the approximate region of the carotid triangles.

10. The controversy over the significance of the relation of lateral vestiges to the stylopharyngeus muscle is critically discussed and a logical interpretation presented.

11. A cartilaginous bar, also termed a cervical or auricular appendage, may well be a second or third branchial arch cartilage vestige unrelated to the pinna anlagen if found in the region of the carotid triangles.

12. An explanation of why branchial cysts are confined to the upper third of the neck, while external or complete fistulae often open externally almost to the sternoclavicular junction is presented.*

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SUBMAXILLARY SALIVARY FLOW: A TEST OF CHORDA TYMPANI NERVE FUNCTION AS AN AID IN DIAGNOSIS AND PROGNOSIS OF FACIAL NERVE PARALYSIS.*

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Disturbances of taste and secretion of saliva are frequently associated with facial nerve paralysis of the peripheral type. The application of these phenomena in localization of the facial nerve lesion is well documented, but the accepted criteria and methods of testing these functions are often variable and unreliable. This is particularly true in testing for chorda tympani function by analysis of taste perception.

The accepted criterion of a functioning chorda tympani nerve is the ability to perceive taste on roughly the anterior two-thirds of the tongue on the corresponding side. In the process of testing, a patient, whose taste is found to be absent by one examiner, may have normal or subnormal taste when tested by another. Inadvertent withdrawal of the tongue results in quick perception by oral and posterior glossal taste buds; also, saliva coming in contact with the solution carries the test substance to the back of the mouth, which further diminishes the reliability of the taste observation.

Guided by the anatomical and physiological fact that facial nerve damage between the geniculate ganglion and the exit of the chorda tympani nerve from the middle ear is accompanied by impairment of chorda tympani nerve function, an attempt was made to find a more objective test which would serve to:

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1. Aid in the clinical localization of the site of nerve damage.

2. Equate the degree of impaired chorda tympani nerve function with the degree of facial nerve paralysis.

3. Aid in measuring and predicting, if possible, spontaneous recovery of the damaged facial nerve.

With a view to accomplish these objectives, this study presents a technique for measuring the salivary secretion of

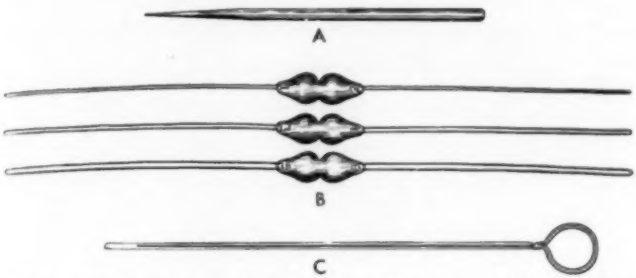

Fig. 1. Apparatus for catheterization of the submaxillary salivary glands, (Wharton's) ducts, include: A. a lacrimal punctum director; B. Bowman's blunt-tipped probes; C. a section of polyethylene tubing, PE No. 60, and a fine wire stylet.

the submaxillary salivary glands as an accurate index of motor function of the chorda tympani nerve.

TECHNIQUE.

The equipment consists of a set of blunt-tipped Bowman lacrimal probes, a punctum director, polyethylene catheters No. 60 (see Fig. 1), and small Kahn type test tubes. The patient is usually placed in the sitting position. A solution of 10 per cent cocaine hydrochloride is applied topically to the orifice of each Wharton's duct. This not only reduces the discomfort associated with dilation, but also makes the orifice visible by stimulating salivary flow. Aseptic technique is used throughout the procedure.

A lacrimal punctum director is inserted into the orifice of

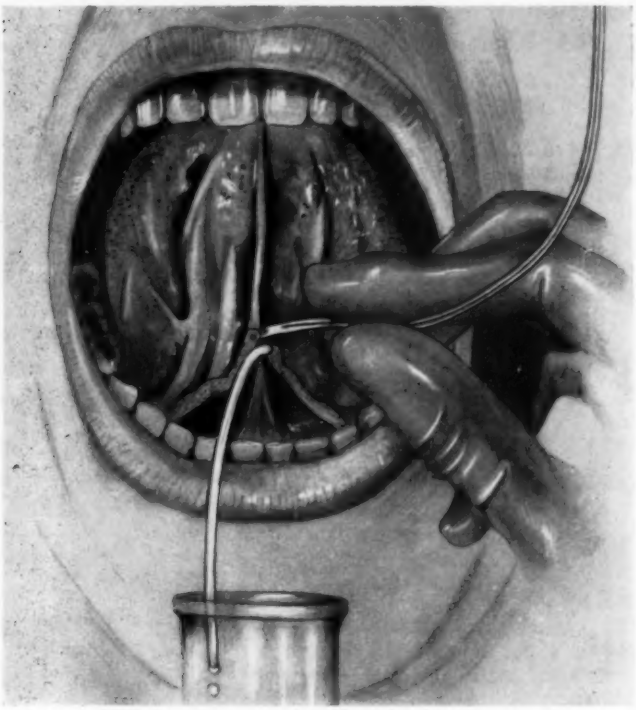

FIG. 2. Catheterization of Wharton's duct and collection of submaxillary gland saliva. An 8 to 10 cm. section of polyethylene tubing is threaded onto a fine rigid wire stylet, and is inserted into Wharton's duct for a distance of 3 to 4 cm. The submaxillary gland saliva is collected in a small Kahn type test tube for each side while the patient sucks on a slice of lemon.

Wharton's duct, after which dilation is accomplished with the Bowman probes, using sizes No. 1 through No. 4. A polyethylene catheter No. 60 (sterilized in 1:500 aqueous benzalkonium chloride for at least 24 hours) is inserted into Wharton's duct for a distance of 3 to 4 cm. This is best accomplished with the aid of a fine rigid wire stylet, onto which an 8 to 10 cm. section of polythylene tubing is threaded.[1] Precaution should be taken to avoid protrusion of the stylet beyond the open end of the polyethylene catheter.

After each Wharton's duct has been catheterized the patient is asked to suck on a slice of lemon for 60 seconds, during which time the output of saliva from each submaxillary gland is collected and measured in drops per minute (see Fig. 2). An assistant collects from one side while the examiner collects from the other. The procedure may be repeated for multiple sampling, after which the catheters are removed and discarded.

MATERIAL FOR STUDY.

We have presumed to add to the lengthy literature on chorda tympani and facial nerve neuropathy because similar patients have been previously studied by the subjective sense, taste. Before presenting our observations it may be well to state something about control tests. In the study of normal individuals between the ages of ten and sixty, the flow of saliva from each submaxillary gland simultaneously is practically of identical volume regardless of the type of stimulus used. For the first five minutes, the quantity is identical; over a 30-minute period the variations between the two salivary glands is not more than 0.50 to 1.0 cc. The volume of saliva produced by lemon stimulation is greater than by chewing paraffin, but in each instance the right submaxillary gland will secrete the same quantity of saliva as the left.[2]

Taste testing for chorda tympani nerve function is also included in this study to illustrate the variations in acuity which produce equivocal results. In testing taste, the tip of the tongue is covered with gauze and held by the examiner until the taste on the anterior two-thirds has been identified by the patient. At this moment the patient points to one of six items written on a card: "taste nothing, taste something but don't know what, bitter, sour, sweet, salty." In all observations, two minutes have been the upper limit for taste determinations in this study. Substances not detected within this time usually will not be appreciated at all with any degree of reliability.

The patients for study have been grouped into the following categories:

I. Cases of chronic suppuration of the middle ear and mastoid.

II. Cases of facial paralysis due to idiopathic Bell's palsy.

III. Cases of facial paralysis associated with skull fractures including the temporal bone.

IV. Cases of facial paralysis associated with central nervous system disease.

GROUP I: CHRONIC SUPPURATION OF THE MIDDLE EAR AND MASTOID.

Case 1: C.A., No. 859083, a 57-year-old mechanic, was admitted on January 4, 1957, to University Hospital for a radical mastoidectomy. There had been drainage from the right ear for approximately 50 years. The patient was unaware of any taste deficiency. On the day prior to operation the following results were obtained in tests for taste and submaxillary saliva:

TASTE.	Right (affected side). Seconds.	Left. Seconds.
Water	Taste Nothing	Taste Nothing
Sweet (50% glucose)	30	30
Sour (vinegar)	30	30
Salty (Hypertonic saline)	30	30
Bitter (quinine)	30	30

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—Min.
Lemon stimulation	42	41

At operation the chorda tympani nerve was found to be intact. The nerve was sacrificed during the radical mastoidectomy. There was no complaint of gustatory deficiency postoperatively. Taste and saliva examinations on the fourth postoperative day and at the end of the second postoperative month were as follows:

TASTE.	Right (affected side). Seconds.	Left. Seconds.
Water	Taste Nothing	Taste Nothing
Sweet	Taste Nothing	30
Sour	Taste Nothing	30
Salty	Taste Nothing	30
Bitter	Taste Nothing	30

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—Min.
Lemon stimulation	0	40

Case 2: G.E., No. 809472, a 45-year-old salesman was operated upon at University Hospital on October 30, 1956, for a chronically draining ear of about 25 years' duration. There was no history of gustatory deficiency. On the day prior to operation the following results were obtained in tests for taste and submaxillary saliva:

TASTE.	Right (affected side). Seconds.	Left. Seconds.
Water	Taste Nothing	Taste Nothing
Sweet	Taste Nothing	30
Sour	Taste Nothing	120
Salty	Taste Nothing	120
Bitter	120	120

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—Min.
Lemon stimulation	0	32

At operation the chorda tympani nerve was found to be absent.

At follow-up examination one month later, the following results were obtained:

TASTE.	Right (affected side). Seconds.	Left. Seconds.
Water	Taste Nothing	Taste Nothing
Sweet	Taste Nothing	Not Recognized
Sour	Taste Nothing	Not Recognized
Salty	Taste Nothing	Not Recognized
Bitter	Taste Nothing	Not Recognized
Bitter	120	120

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—Min.
Lemon stimulation	0	42

DISCUSSION.

Taste testing in these cases was equivocal. In another patient in this group, the salivary flow on the affected side was reduced 50 per cent when compared with the normal side. At operation, the chorda tympani was found imbedded in scar and granulation tissue. In this particular patient preoperative testing of taste was highly equivocal, and no decision could be reached as to whether taste was present. Thus partial function of the chorda tympani nerve may also be detected by comparing submaxillary salivary secretion from both glands.

The first patient (C.A.) presented is characteristic of the individual who has an intact chorda tympani nerve prior to operation, which must necessarily be sacrificed in the performance of a radical mastoidectomy. The paralytic effect of chorda tympani nerve section upon submaxillary salivary flow is illustrated.

The second patient (G.E.) also demonstrates the difficulty

encountered in eliciting a consistent response on the normal side. It is of interest to note the reaction to bitter (quinine) in both cases. The delay in identifying bitter taste in the second patient probably indicates that the test solution had travelled to the posterior one-third of the tongue, where practically all the gustatory receptors for bitter taste are located. That there was no complaint of gustatory deficiency after recent complete removal of the chorda tympani nerve (Case 1) and after long term destruction of it (Case 2) appears to substantiate the observation of Crosby.[3] She states that although modalities of taste can be recognized at subcortical levels, an individual's higher (cortical) centers are usually not concerned with the analysis of taste impressions from a specific geographical area on the tongue or palate; rather, the combination of taste, smell and texture, experienced during eating, are cortical associations which are acquired during the individual's own experience. If a disturbance of taste does exist, the difficulty in localization may cause the patient to become confused and thus complain that a portion of the tongue feels "numb."

The second patient (G.E.) demonstrates the loss of chorda tympani function in long standing middle ear suppuration. There is no flow of saliva by reflex chemical stimulation (sucking a lemon) preoperatively and postoperatively. This phenomenon, observed in 11 similar patients in this Clinic, challenges the concept of reflex formation of saliva in the absence of secretory (motor) innervation—the so-called paralytic secretion of saliva. In this instance absence of salivary flow exists after possibly two decades of chorda tympani destruction!

GROUP II: FACIAL PARALYSIS DUE TO IDIOPATHIC BELL'S PALSY.

Case 1: H.W., No. 37565, a 53-year-old telephone lineman, was admitted to St. Joseph Mercy Hospital on February 15, 1956, because of inability to close the left eye and weakness of the left corner of the mouth for 24 hours. On the day prior to admission the patient had been working in the rain.

Physical examination revealed a blood pressure of 180/100 (RAR); the patient could not move the corner of the mouth or wrinkle the left side of the nose. The left eye could be partially closed; there was no difficulty in wrinkling the forehead.

The neurological consultant felt that the patient exhibited a central

type of facial paresis, probably compatible with an hypertensive encephalopathy, without other neurological deficit. Further examination noted that the patient could not smile on command or by emotional expression. The hearing was normal. Roentgenograms of the skull were normal. The following results were obtained for taste and saliva tests:

TASTE.	Left (affected side). Seconds.	Right. Seconds.
Water	Taste Nothing	Taste Nothing
Sweet	Not Recognized	Not Recognized
Sour	Not Recognized	Not Recognized
Salty	Not Recognized	Not Recognized
Bitter	Not Recognized	Not Recognized

SALIVA.	Left (affected side). Drops—Min.	Right. Drops—Min.
Lemon stimulation	14	20

On the fourth hospital day there was a complete left peripheral facial paralysis without other neurological deficit. Taste and saliva examination was as follows:

TASTE.	Left (affected side). Seconds.	Right. Seconds.
Water	Taste Nothing	Taste Nothing
Sweet	Taste Nothing	Not Recognized
Sour	Taste Nothing	Not Recognized
Salty	Taste Nothing	Not Recognized
Bitter	Taste Nothing	120

SALIVA.	Left (affected side). Drops—Min.	Right. Drops—Min.
Lemon stimulation	0	25

The patient was discharged on the fifth hospital day with the diagnosis of Bell's palsy. One week later (13 days after the onset of the facial paralysis) there was partial motion of all the muscles of facial expression except forehead motion, which was completely absent. Taste and saliva test results were as follows:

TASTE.	Left (affected side). Seconds.	Right. Seconds.
Water	Taste Nothing	Taste Nothing
Sweet	Not Recognized	Not Recognized
Sour	Not Recognized	Not Recognized
Salty	Not Recognized	Not Recoginzed
Bitter	Not Recognized	Not Recognized

SALIVA.	Left (affected side). Drops—Min.	Right. Drops—Min.
Lemon stimulation	15	30

Eight weeks after the onset of facial paralysis, there was complete return of function. The results of taste examination were identical to those of the first test. Submaxillary saliva examination results were as follows:

SALIVA.	Left (affected side). Drops—Min.	Right. Drops—Min.
Lemon stimulation	50	49

Case 2: B.L., No. 805312, a 32-year-old white housewife, suddenly developed weakness of the left corner of the mouth during an upper respiratory infection. Twenty-four hours after onset, there was a complete left facial paralysis. On February 15, 1955, two days after the onset, the patient visited the Otolaryngology Clinic at University Hospital. She complained of a deficiency of taste on the left side of the tongue. Loud noises had an "echoing quality," and she did not wish to listen to radio or television because the sound "hurt" her ears, particularly the left one. The physical examination revealed a complete left peripheral type of facial paralysis without other neurological deficit. A pure tone audiogram showed normal hearing. The tuning fork and Tschiassny stethoscope tests for phonophobia were positive in the left ear. Roentgenograms of the mastoid bones revealed no abnormalities. The following results were obtained for taste and saliva tests:

TASTE.	Left (affected side). Seconds.	Right. Seconds.
Water	Taste Nothing	Not Recognized
Sweet	Not Recognized	Instantly
Sour	Not Recognized	Instantly
Salty	Not Recognized	Instantly
Bitter	Not Recognized	Instantly

SALIVA.	Left (affected side). Drops—Min.	Right. Drops—Min.
Lemon stimulation	29	43

On the thirty-fifth day after onset of facial paralysis, the patient showed virtually complete return of facial function except for a slight weakness of the forehead. There were no complaints of phonophobia or gustatory deficiency. The following results were obtained for taste and saliva tests:

TASTE.	Left (affected side). Seconds.	Right. Seconds.
Water	Recognized as Sweet	Taste Nothing
Sweet	30	Instantly
Sour	30	Instantly
Salty	"Sour or Bitter"	"Sour or Bitter"
Bitter	30	Instantly

SALIVA.	Left (affected side). Drops—Min.	Right. Drops—Min.
Lemon stimulation	56	54

Forty-five days after onset of facial paralysis there was complete return of facial movements.

Case 3: D.S., No. 41361, a 32-year-old mechanic, noted weakness of the right corner of the mouth on arising in the morning. Twenty-four hours later, on May 10, 1956, there was tearing and difficulty in closing the right eye. No gustatory deficiency was noted. Physical examination revealed a complete right peripheral type of facial paralysis without other neurological deficiencies. Otologic studies showed a conductive hearing loss of long standing, compatible with clinical otosclerosis. Roentgenograms of the mastoids revealed no abnormalities. The following results were obtained for taste and saliva tests:

TASTE.	Right (affected side). Seconds.	Left. Seconds.
Water	Taste Nothing	Taste Nothing
Sweet	Taste Nothing	30
Sour	Taste Nothing	Instantly
Salty	Taste Nothing	Instantly
Bitter	Taste Nothing	Instantly

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—Min.
Lemon stimulation	9	35

On May 15, 1956, five days later, there was no change in the facial paralysis. The following results were obtained for taste and saliva tests:

TASTE.	Right (affected side). Seconds.	Left. Seconds.
Water	Taste Nothing	Taste Nothing
Sweet	Not Recognized	Instantly
Sour	Not Recognized	Instantly
Salty	Not Recognized	Instantly
Bitter	120	Instantly

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—Min.
Lemon stimulation	28	40

Eighteen days later the patient showed considerable return of facial nerve function. Only the frontalis muscle showed minimal activity. Tests for taste and saliva were as follows:

TASTE.	Right (affected side). Seconds.	Left. Seconds.
Water	Taste Nothing	Taste Nothing
Sweet	Not Recognized	Instantly
Sour	Not Recognized	Instantly
Salty	Not Recognized	Instantly
Bitter	Not Recognized	Instantly

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—min.
Lemon stimulation	37	40

Twenty-four days after the onset of facial paralysis there was complete return of facial movement.

Case 4: E.T., No. 820213, a 48-year-old colored housewife, complained of inability to move the right side of the face for the past 24 hours. Physical examination on August 18, 1955, revealed a complete right peripheral type of facial paralysis without other neurological deficit. Otologic testing showed normal hearing. The tuning fork and Tschiassny stethoscope tests for phonophobia were positive in the right ear. The following results were obtained for taste and saliva tests:

TASTE.	Right (affected side). Seconds.	Left. Seconds.
Water	Taste Nothing	Taste Nothing
Sweet	Taste Nothing	Instantly
Sour	Taste Nothing	Instantly
Salty	Taste Nothing	Instantly
Bitter	30	Instantly

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—Min.
Lemon stimulation	0	32

One month later, there was no improvement of the facial paralysis, and a lateral band tarsorrhaphy was performed for prevention of exposure keratitis. Submaxillary salivary secretion tests revealed tne following:

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—min.
Lemon stimulation	0	40

The patient did not return until January, 1956, six months after onset of the facial paralysis. At this time there was residual weakness of all the facial musculature on the right side. The following tests were obtained for taste and saliva:

TASTE.	Right (affected side). Seconds.	Left. Seconds.
Water	Taste Nothing	Taste Nothing
Sweet	Not Recognized	Instantly
Sour	Not Recognized	Instantly
Salty	Not Recognized	Instantly
Bitter	60	Instantly

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—min.
Lemon stimulation	19	42

The patient failed to appear at regular intervals and was not seen again until November, 1957, twenty-seven months after onset of facial paralysis. There was residual weakness of all the facial musculature but sufficient orbicularis oculis function to permit removal of the tarsorrhaphy. The following results were obtained for taste and saliva tests:

TASTE.	Right (affected side). Seconds.	Left. Seconds.
Water	Taste Nothing	Taste Nothing
Sweet	Not Recognized	60
Sour	Not Recognized	Instantly
Salty	Not Recognized	Instantly
Bitter	60	Instantly

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—min.
Lemon stimulation	13	35

DISCUSSION.

Ten patients with Bell's palsy were studied in this investigation. Four have been selected for presentation because they demonstrate certain refinements in diagnosis of facial nerve disease which heretofore have not been reported.

The first patient (H.W.) presents the complete clinical picture of Bell's palsy from its inception. Such a patient, seen

early in the course of facial paralysis, may be erroneously diagnosed as a central type of facial paralysis; however, submaxillary salivary testing of chorda tympani nerve function was helpful in differentiating central from the peripheral type of facial paralysis. The fact that the chorda tympani nerve was impaired, though functioning, is evidence of a peripheral rather than a central lesion. In this instance it was also apparent that the degree of impaired chorda tympani nerve function could be correlated with the degree of facial nerve paralysis. In this instance the recovery of chorda tympani nerve function corresponds with facial nerve recovery.

The last three patients of this group demonstrate the more common situation observed when the patient consults a physician for a condition which turns out to be a complete unilateral peripheral facial paralysis due to Bell's palsy. It is of note that such a patient may demonstrate partial chorda tympani nerve activity by salivary testing, in the presence of a complete facial paralysis. This observation suggests the possibility that the patient may be spared the sequelae of imperfect facial nerve recovery. It may justify also the prediction of a favorable prognosis for spontaneous recovery from complete peripheral facial paralysis when it can be demonstrated that the chorda tympani nerve retains its function or rapidly becomes functional early in the course of Bell's palsy. It would appear also that the involvement of the facial nerve, by whatever pathologic process in Bell's palsy, is not severe, if the chorda tympani nerve is partially spared. Further study is necessary, however, if these assumptions are to be confirmed.

The fourth patient (E.T.) demonstrates the sequelae of imperfect recovery and facial disfigurement. The partial return of facial nerve and chorda tympani nerve function appears to correlate for a two-year period. Since the rate of chorda tympani nerve recovery did not exceed that of the facial nerve, the prognosis for facial nerve repair, in retrospect, appears to have been poor.

In this group of patients we note the variability and un-

reliability of taste testing in situations where there is impaired function of the chorda tympani nerve and facial nerve with subsequent spontaneous recovery of both. In the third patient (D.S.) absence of taste was recorded in the presence of minimal but definite chorda tympani nerve activity as evidenced by submaxillary salivary flow on lemon stimulation.

The fourth patient (E.T.) demonstrates another reason for the unreliability of taste testing on the anterior two-thirds of the tongue. Engström and Wohlfart[4] state that in embryological development some fibers of the glossopharyngeal nerve may be drawn forward in the tongue and serve to conduct gustatory impulses in the absence of chorda tympani innervation. This concept may be substantiated by the fact that bitter taste was the only definite gustatory experience unequivocally identified by this patient.

GROUP III: FACIAL PARALYSIS ASSOCIATED WITH SKULL FRACTURES.

Case 1: J.B., No. 807846, a 20-year-old student was admitted to University Hospital on April 8, 1955, for a severe head injury sustained in an automobile accident. Physical examination revealed a complete right peripheral type of facial paralysis, conductive deafness and hemotympanum. Roentgenograms revealed a slight tempor-parietal fracture which extended into the petrous ridge toward the medial portion of the internal auditory canal. Taste and saliva studies performed on April 18, 1955, were as follows:

TASTE.	Right (affected side). Seconds.	Left. Seconds.
Water	Taste Nothing	Taste Nothing
Sweet	Taste Nothing	Instantly
Sour	Taste Nothing	Instantly
Salty	Taste Nothing	Instantly
Bitter	Taste Nothing	Instantly

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—Min.
Lemon stimulation	6	68

Three weeks after the accident a complete facial paralysis was still present. Taste examination was unchanged. The submaxillary salivary studies were as follows:

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—Min.
Lemon stimulation	12	55

Six weeks after injury the hearing had returned to normal. There was some motion of the forehead and the upper lip on the right side. Taste and saliva studies were as follows:

TASTE.	Right (affected side). Seconds.	Left. Seconds.
Water	Taste Nothing	Taste Nothing
Sweet	Not Recognized	Instantly
Sour	Not Recognized	Instantly
Salty	Not Recognized	Instantly
Bitter	Not Recognized	Instantly

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—Min.
Lemon stimulation	20	65

Four months after injury the patient could close the right eye almost completely, had good symmetry of the face in repose, but the motion of the forehead was not noticeably improved. Taste examination was the same as performed six weeks after injury. Saliva examination was as follows:

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—Min.
Lemon stimulation	40	60

The patient subsequently left Michigan and has not been heard from since.

Case 2: R.K., No. 805674, a 23-year-old male student, was admitted to University Hospital on March 2, 1955, because of a right facial nerve paresis. On February 13, 1955, he had sustained a severe head injury with a linear fracture in the right temporal region from an automobile accident. Physical examination revealed a right-sided weakness of all the facial muscles of expression, the muscles of the forehead being the most severely impaired. Otologic examination showed a hemotympanum and a conductive deafness, A.D. Roentgenograms revealed a fracture through the occipital bone, occipito-temporal suture, extending into the petrous portion of the temporal bone.

Taste and saliva studies were as follows:

TASTE.	Right (affected side). Seconds.	Left. Seconds.
Water	Taste Nothing	Taste Nothing
Sweet	Not Recognized	Instantly
Sour	120	Instantly
Salty	120	Instantly
Bitter	120	Instantly

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—Min.
Lemon stimulation	32	58

Three weeks later facial mobility was much improved. Taste examination was the same as performed previously. Saliva examination was as follows:

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—Min.
Lemon stimulation	86	105

Nine weeks after injury, the return of facial movements was complete except for minimal frontalis weakness. Taste and saliva studies were as follows:

TASTE.	Right (affected side). Seconds.	Left. Seconds.
Water	Taste Nothing	Taste Nothing
Sweet	30	Instantly
Sour	30	Instantly
Salty	30	Instantly
Bitter	30	Instantly

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—Min.
Lemon stimulation	70	72

Case 3: G.S., No. 854039, a 28-year-old welder, was transferred to University Hospital on October 3, 1956. On September 29, 1956, the patient was involved in an automobile accident and sustained a severe head injury and fractured mandible. Physical examination revealed a complete right peripheral type of facial paralysis. There was an extensive keratitis in the right eye. A deformity of the superior portion of the external auditory canal prevented adequate visualization of the right tympanic membrane. There was a conductive deafness on the right side. Roentgenograms revealed a fracture of the mandible and a temporo-parietal fracture with extension through the osseous right external auditory canal.

Taste studies were omitted because of difficulty in protruding the tongue. Submaxillary salivary examination was as follows:

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—Min.
Lemon stimulation	0	52

On October 8, 1956, decompression and neurolysis of the facial nerve was performed through a modified radical mastoidectomy approach. Open reduction of the mandible with internal fixation and a lateral band tarsorrhaphy were subsequently performed.

In February, 1957, four postoperative months, there was only minimal motion of the upper lip and nasal ala. Salivary studies were as follows:

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—Min.
Lemon stimulation	9	65

In October, 1957, one year after facial nerve decompression, minimal motion of the upper and lower lips and side of the nose was present. Submaxillary salivary testing showed the following:

SALIVA.	Right (affected side). Drops—Min.	Left. Drops—min.
Lemon stimulation	4	70

DISCUSSION.

Six patients demonstrating facial nerve paralysis associated with skull and temporal bone fractures comprise this facial nerve trauma group. Three cases have been described to

PRINCIPAL ORGANIC AND INORGANIC CONSTITUENTS OF PURE SUBMAXILLARY SALIVA.
(LEMON STIMULATION)

STUDIES	PATIENT—B.L., Case 2, Group II.				PATIENT—R.K., Case 2, Group III.			
	Involved Side		Normal Side		Involved Side		Normal Side	
	At Initial Visit	At Recovery	At Initial Visit	At Recovery	At Initial Visit	At Recovery	At Initial Visit	At Recovery
pH	7.35	7.4	7.5	7.45	7.52	7.5	7.62	7.55
Amylase (units)	28,900	27,500	30,000	28,000	23,600	23,900	40,200	41,000
Protein (gm. %)	0.11	0.04	0.2	0.18	0.012	0.009	0.16	0.15
Creatinine (mg. %)	0.13	0.18	0.11	0.16	0.35	0.35	0.35	0.30
Uric Acid (mg. %)	1.4	1.1	1.5	1.4	1.0	0.8	1.1	1.4
Sodium (meq/L)	16	24	24.8	26	22	27	32.9	36
Potassium (meq/L)	18	16	13.3	15	18	15	17	20
Chloride (meq/L)	14.3	18.1	28	31	trace	trace	22	21
Bicarbonate (as CO_2 c.p. in meq/L)	12.3	11.7	15	16.8	17.7	23.1	10.4	11.7
Calcium (mg. %)	3.4	4.1	2.1	3.2	5.0	4.3	4.2	4.3
Phosphorus (mg. %)	5.6	5.0	3.6	4.1	5.9	4.2	4.5	4.5
Total Volume Collected	18ml/45min.	30ml/45min.	28ml/45min.	29ml/45min.	22ml/45min.	65ml/45min.	63ml/45min.	65ml/45min.

Fig. 3. The principal organic and inorganic constituents of pure submaxillary saliva from patients with peripheral facial paralysis associated with Bell's palsy and skull fractures who have recovered full facial nerve function spontaneously. The saliva was collected in individual containers via polyethylene catheters using lemon stimulation. The values obtained for the various constituents do not vary significantly in spite of the diminished volume of secretion, thus demonstrating the "all or none" principle of nerve impulse transmission in the preganglionic parasympathetic fibers of the chorda tympani nerve.

emphasize certain clinical and neuro-anatomical observations pertaining to chorda tympani nerve testing.

As noted in the Bell's palsy group, it appears that damage of the facial nerve due to trauma may not be severe if the chorda tympani nerve is partially spared. (Obviously extra-temporal injury of the facial nerve will not involve the chorda tympani nerve). Since chorda tympani nerve function is present in the first (J.B.) and second (R.K.) patients, it seemed in retrospect to indicate potential spontaneous recovery of good facial nerve function. That recovery of both the chorda tympani nerve and the facial nerve occurred bears this out.

The second patient (R.K.) illustrates the value of chorda tympani testing as an aid in localization of the facial nerve lesion in partial as well as complete facial paralysis.

In the third patient (G.S.) the absence of chorda tympani nerve, as well as facial nerve function, were factors in making a decision for immediate decompression.

Figure 3 is a quantitative tabulation of the principal organic and inorganic constituents of pure submaxillary saliva in patients with Bell's palsy and facial paralysis associated with temporal bone fractures who have recovered full facial nerve function spontaneously. The "all or none" principle of nerve impulse transmission in the preganglionic parasympathetic fibers of the chorda tympani (the visceral motor fibers responsible for submaxillary salivary secretion) is demonstrated. It would appear from the three cases illustrated that the constituents of pure submaxillary saliva, obtained by polyethylene catheterization of Wharton's ducts are not much changed quantitatively even though the salivary secretion is quantitatively diminished.

GROUP IV: FACIAL APARALYSIS ASSOCIATED WITH CENTRAL NERVOUS SYSTEM DISEASE.

Report of Case: M.F., No. 552842, a 58-year-old housewife visited the Neurology Clinic of University Hospital on May 24, 1955, complaining of weakness of the right corner of the mouth of one week's duration. There was no antecedent history of upper respiratory infection or of hypertension. The patient stated that she awoke in the morning to find a great

deal of difficulty in moving the right corner of the mouth and that it seemed to droop.

Examination by the neurologists revealed a right-handed white female with a blood pressure of 150/100 (RAR). There was a lower facial paresis with poor delineation of the naso-labial fold. Minimal impairment of frontalis movement was also found. There was no other neurological deficit. The clinical impression was Bell's palsy.

Otolaryngological consultation on May 26, 1955, revealed only a lower facial paresis. Audiometric studies showed presbycusis. Roentgenograms of the skull were normal. Taste and saliva studies were as follows:

TASTE.	Right (affected side). Seconds.	Left. Seconds.
Water	Taste Nothing	Taste Nothing
Sweet	30	30
Sour	30	30
Salty	30	30
Bitter	30	30
SALIVA.	Right (affected side). Drops—Min.	Left. Drops—Min.
Lemon stimulation	20	20

Two weeks after onset of facial paresis there was minimal weakness of the right upper and lower lips. Taste and saliva studies revealed no change from similar testing performed on May 26, 1955.

Seven months after the patient's initial visit there was complete facial symmetry. Taste and saliva studies were consistent with those performed at the time of the first otolaryngological consultation. Final diagnosis: Mild hypertensive encephalopathy.

DISCUSSION.

Three cases comprised this group: a patient with hypertensive encephalopathy; second, a patient with a nuclear lesion of the VIIth cranial nerve due to bulbar poliomyelitis, and a patient with a brain tumor in the floor of the fourth ventricle. Other patients with obvious central facial paralysis were studied for documentary purposes only. Indeed, the presence of equal submaxillary salivary flow on lemon stimulation indicating an intact chorda tympani nerve is another diagnostic aid in localizing a facial nerve lesion.

SUMMARY.

A clinical test for chorda tympani nerve function by measuring submaxillary salivary secretion is presented as a practical office procedure. The technique of submaxillary (Wharton's) duct catheterization is described. It has been demon-

strated that this procedure is a far more accurate index of chorda tympani nerve function than are tests for taste.

Chorda tympani nerve function by measurements of submaxillary salivary gland secretion can serve as an aid in the clinical localization of facial nerve damage. The peripheral and central types of facial paralysis can be determined when such differentiation is not obvious.

In the peripheral type of facial paralysis, submaxillary salivary testing of chorda tympani nerve function appears to be a consistently reliable adjunct for intra and extra temporal localization of facial nerve injury.

The degree of impaired chorda tympani function can be correlated with that of facial nerve paralysis when both are involved. A good prognosis for spontaneous recovery might be predicted if it can be demonstrated that the chorda tympani nerve retains its function early or becomes functional rapidly in the course of Bell's palsy or traumatic facial nerve injury; however, further studies are necessary to substantiate this observation.

The chorda tympani nerve contains the pre-ganglionic secretory fibers which activate the submaxillary ganglion and other parasympathetic ganglion cells imbedded in the substance of the submaxillary gland. If the chorda tympani nerve is destroyed, no submaxillary salivary secretion occurs when the gland is stimulated by reflex chemical stimulation (eating) because the secretory fibers have been removed. This observation challenges the concept of reflex formation of saliva in the absence of secretory innervation—the so-called paralytic secretion of saliva.

Taste perception as an index of chorda tympani function is variable, and in many instances unreliable in testing the normal and abnormal sides of the tongue in conditions where there is simultaneous impaired function of the chorda tympani nerve and facial nerve.*

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POST GRADUATE COURSES AT TEMPLE UNIVERSITY SCHOOL OF MEDICINE AND HOSPITAL.

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DIAGNOSIS AND MANAGEMENT OF CHEMICAL BURNS OF THE ESOPHAGUS.*

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Ingestion of a corrosive substance creates a very difficult problem, both to the unfortunate patient and the attending physician, in treatment of the damaged esophagus. Solution of this problem has been sought since the time of Pliny, when it is recorded that the ashes of plants were treated with lime to produce sodium and potassium hydroxide for use in the manufacture of soap.

It is the purpose of this manuscript to illustrate how the treatment of many of the suspected cases of esophageal burns can easily be resolved, and to correlate a rational regimen for the management of all cases suspected of ingesting a corrosive chemical.

From January, 1952, to the present time esophagoscopy has been done on all patients presenting themselves at Children's Hospital of Michigan with a history of ingestion, or with evidence of fresh burns of the lips, tongue, mouth or pharynx, within the first ten days of contact with the chemical. This procedure was accomplished against moderate opposition from some of the endoscopic staff, who felt that esophagoscopic examination at this period carried considerable risk. It was also suggested that a damaging burn might escape notice through the esophagoscope. Both of these fears proved to be without foundation; therefore, the case study to be presented represents 51 patients admitted, for examination and treatment from January, 1952, through July, 1957, to a hospital which limits its admissions to children under 13 years of age.

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ETIOLOGY AND AGE.

Of the 51 cases, 33 had a history of contact with lye, and eight with ingestion of commercial preparations under the trade name of Drano[7] and Sani-Flush.[1] These commercial preparations are composed approximately of 95 per cent sodium and potassium hydroxide. Another which should be included in this group was from potash used to prepare a "hair oil mixture." Four cases were a result of drinking Lysol, two from household ammonia water, and one each from cresylic acid, phenol, and potassium permanganate (see Table I).

TABLE I.

Agent	No. of Cases	Degree of Burn (Esophagoscopy) None	Mild (Erythema)	Moderate
Lye	33	21	5	7
Drano	7	4	2	1
Sani-Flush	1		1	
Potash	1	1		
Lysol	4	4		
Ammonia Water	2	2		
Potass. Permanganate	1	1		
Phenol	1		1	
Cresylic Acid	1	1		
Total	51	34	9	8

Although evidence of mouth burns was present in all nine cases of chemical ingestion not in the alkali group, no burns were detected by esophagoscopy except one from phenol, which was classified as mild and required no treatment. This finding would agree with Owens,[1] in his report of 131 cases of chemical burns, in which he concluded that "ingestion of household chemicals, other than lye, in general use today need not receive the drastic treatment given the latter, as stricture formation from their ingestion is a rarity."

All 51 patients were five years of age, or under. Thirty-two were two years of age or under, and 19 were from three to five years of age (see Table II). This finding, in a hospital which admits patients to the age of 13, would indicate that most of these accidents occur in children who are not old enough to differentiate poisons from the usual food and

drink. The seven-months-old baby was accidentally given phenol, which had been used as a toothache remedy by the family. Another chewed a potassium permanganate tablet mistaken for candy. Some ate from cans of lye crystals left open on the floor. Others drank a solution of lye from bottles.

From these findings, it is obvious that most of these accidental burns are due to the careless accessibility of open cans of lye to children, the placing of caustics in containers used for food (cup, glass, or soda pop bottle), or medicine cabinet accidents. Lye in solution is a whitish, cloudy mixture which can easily be mistaken for milk by a child.

Holinger and Johnston,[2] in their study of 96 cases during a 13-year period, found in the five to ten year age group, one

TABLE II.

Age	No. of Cases
7 months to 2 years	32
3 years to 5 years	19
6 years to 12 years	0

who drank lye on a "dare," and another who drank muriatic acid from a coke bottle. In the 11 to 20 age group, all were females, most of whom drank a corrosive chemical because of disappointment in love. Over age 20 were those with psychopathic personalities intent on suicide.

All of the cases in the present study can be considered accidental, since they were age five or under, and a social service worker examined the homes and carefully questioned the parents when any possible suspicious circumstance was encountered. In none was there found any evidence of homicidal intent.

PATHOLOGY.

As previously stated, lye in its pure form, or in the commercial products where it is present in high concentrations, is by far the most damaging agent. In the present series, none of the other chemicals caused significant changes in the esophagus. Other reports in the literature contain cases of

scarring from phenol, formaldehyde, ammonia water, Lysol, sulfuric acid, nitric acid, lactic acid, potassium permanganate, iodine and bichloride of mercury, but it is admitted that these do not usually cause strictures.

Alkalies are the most destructive because of their ability to penetrate deeply into the layers of the esophagus to produce a liquifying necrosis. Acids cause less destruction because they cause a coagulation of the superficial layers, forming a firm eschar which limits penetration to the deeper layers. Destruction of the stomach is apt to be much more significant from acids. The others, such as Lysol, may produce a mucosal surface erythema or superficial esophagitis, but the healing is rapid without sufficient scar to form strictures; therefore, this discussion of pathologic changes in the esophagus will be limited to ingested caustics.

The relationship between the depth of penetration of the lye, and its concentration, has been shown in experimental animals by Krey,[3] who examined the effects of a ten-~~minute~~ second contact of different strengths of sodium hydroxide on the rabbit's esophagus. He found that a N-solution (3.8 per cent) produced necrosis of the mucosa and submucosa, with some involvement of the inner longitudinal layers. With a 3 N-solution (10.7 per cent), the necrosis extended to the circular muscle. A 5 N-solution (16.4 per cent) involved the outer longitudinal muscle. Necrosis extended throughout the esophageal wall with a 7 N-solution (22.5 per cent), and some effect occurred also to the periesophageal tissues. These findings are significant when it is considered that ordinary washing powders may contain a concentration of caustic soda in excess of 7 N-solution; also, it is obvious that only small quantities of a concentrated caustic, mixed with the saliva and swallowed, may produce severe lesions of the esophagus. Larger quantities of a more dilute solution, as in a suicidal or homicidal attempt, may cause not only severe trauma to the esophagus, but the stomach and duodenum are also in danger of involvement.

The histopathologic tissue changes will depend upon the depth of penetration:

1. A superficial burn may be represented by only a surface erythema, or there may be destruction of areas of surface mucosa without damage to the deeper tissues. These areas will heal without scar formation, and there need be no fear of a stricture.

2. A deeper burn will penetrate the submucosal and muscular layers, causing a desquamation of the mucosa, leaving these underlying layers exposed. Belinoff[4] divided the course of this degree burn into the four stages of necrosis, ulceration, granulation, and scar formation, from observations on the experimentally-burned dog's esophagus. The tissue cells of the submucosal coats are destroyed by coagulation of the intracellular proteins, and an intense inflammatory reaction is set up. All layers of the esophagus have become involved in this acute inflammation during the first week, and the organ becomes semi-rigid. The inflammation also involves the myenteric plexus of nerves, including both Meisner's and Auerbach's, so that both the muscles and nerves are involved in the degenerative process. During this stage, the patient is seriously ill and is frequently unable to swallow his own saliva.

Following this, new blood vessels begin to form with the proliferation of numerous fibroblasts. By the end of the second week, collagen fibers have been laid down by the fibroblasts to fill the defect. Extensive granulations form with new blood vessels freely ramifying through this newly formed connective tissue. At this time, the swallowing function returns approximately to normal, and the patient may be mistakenly believed to be out of trouble.

Beginning about the third week, the collagen fibers begin to contract. Adhesions, forming between the granulating areas, form bands and pockets with gradual narrowing of the esophagus. As this contraction continues over a period of several months, the return of dysphagia may be apparent only in a change of the patient's eating habits; he will eat fewer solids, washing down each mouthful with water or milk, and he will learn to gag himself to raise food when it becomes impacted.

When healing is finally accomplished, a dense fibrous coat has replaced the submucosa and muscular layers, and the newly formed epithelium is of a low stratified type without glands. This end-result consists of an esophageal lumen with many pockets and channels, or the lumen may be completely obliterated.

3. A still deeper penetration will cause necrosis through the entire wall of the esophagus causing a periesophagitis, and may even result in perforation with mediastinitis, pericarditis, or broncho-esophageal fistula. A burn of this severity may not allow the patient to live through the stages of contraction and stricture formation. Blassingame, et al.,[5] list early deaths as being due to severe pulmonary edema from aspiration of lye solution, massive necrotizing esophagitis, or severe shock.

SIGNS AND SYMPTOMS.

Marked edema and redness is evident immediately on the parts contacted by the corrosive. The lower lip is usually involved to the greatest degree, with the mouth, tongue and pharynx being involved to a lesser extent. A white membrane forms very rapidly in the more severely burned areas. Immediate dysphagia occurs, accompanied by profuse salivation, which lasts from two or three days to a week.

DIAGNOSIS.

Every patient with a history of ingestion, or evidence exhibited by burns of the lips, mouth, or pharynx from contact with a corrosive, must be considered to have an esophageal burn until proven otherwise. A dilute solution may not show mouth burns, because of an extra heavy coating of mucous and saliva at the time of the ingestion, but still cause damage to the delicate esophageal membrane; therefore, suspicion of a burn of the esophagus should be aroused on history alone. It was felt by the author that many of these patients with presumptive evidence of ingestion of a corrosive, did not suffer significant esophageal damage, and for this reason the 51 patients included in this study were esophagoscoped

from five to ten days after the accident. It was felt that this was the only way of making a *positive diagnosis* of an esophageal burn, other than waiting for the formation of a stricture.

Many authors have condemned early esophagoscopy because of the supposed hazard of perforation. Others recommend delaying the procedure for two to three weeks (Jackson,[6] Woodward[7]). Lederer[8] advises that esophagoscopic examination is indicated only in "those patients who have no burns in the mouth or pharynx and when the agent is not known." Hanckel[9] states that esophagoscopy should be done in all cases, but not for several weeks after the burn has occurred in order to avoid perforation.

Analysis of the 51 cases in this study has proven esophagoscopy to be a safe procedure, when done during the first five

TABLE III.

Physical Findings	No. of Cases	Degree of Burn (Esophagoscopy) None	Mild (Erythema)	Moderate
Burns of Lip, Mouth, Tongue, or Pharynx	43	27	9	7
No Visible Burns External	8	7	0	1

to ten days, in that there were no complications from the procedure in a single instance; and most of the examinations were done by the resident, in training, under competent staff supervision. The value of such a procedure is shown in Table III, where 34 patients had no evidence of esophageal burn, nine had areas of mild erythema which required no treatment, and only eight demonstrated a burn requiring follow-up treatment. Many unnecessary and unpleasant treatments by dilatation were thus avoided by 43 patients, much valuable time by the medical staff was preserved, and several extra days of expensive hospitalization and precious beds were saved.

It is granted that there has not been enough lapse of time in the more recent cases to be sure that the untreated cases may not develop a narrowing of the esophagus at a future date. Stothers[10] mentions a case of sudden acute impaction occurring 40 years after the accident; however, follow-up

has been done in most cases since the beginning of the study in 1952, by repeated barium examinations, and none to the present time has shown evidence of esophageal narrowing. If any of these patients should develop dysphagia in the future, it is safe to assume that the narrowing would be of the semi-lunar or "shelf type," which would respond readily to a few dilatations.

It is also demonstrated in Table III that the long prevalent assumption that lye burns of the lips, tongue, mouth and pharynx usually infer esophageal damage is false, since only eight out of 43 patients were found to have burns of the esophagus requiring treatment. These findings are even more startling than those of Cody[11] who reports a series of 23 cases of mouth burns in which only 12 demonstrated esophageal pathology. From these data it is obvious that a false impression may have been presented by many of the reports in the literature of the prevention of stricture formation by early dilatation, since a large percentage of these patients undoubtedly had no esophageal damage at the begininng.

The findings in this case study would agree with Stumboff,[12] who asserts that esophagoscopy can be safely performed during the first stage of an essophageal burn "because the wall of the esophagus is covered with a thick membrane;" and he doesn't feel that it is a safe procedure later during the presence of ulcerations and granulations. Daly[13] warns against esophagoscopy in the third week, when scar tissue contraction has begun. This belief is supported by one of our cases, when there were no complications following diagnostic esophagoscopy done eight days after the ingestion of lye, in which a severe burn was demonstrated; however, an ill-advised esophagosocpy and dilatation two weeks later resulted in perforation, followed the next day by air and exudate in the right pleural space and mediastinitis, and death within two days.

It has been our practice to use general anesthesia to reduce risk of trauma, and a careful examination of the hypopharynx is done before entering the esophagus. If a burn is found in this area, the esophagoscope is carefully introduced through

the cricopharyngeus and advance of the instrument is immediately halted at the first sign of damage except in the presence of only mucosal erythema. In case of the latter, the examination is continued until signs of actual penetration are encountered.

Under these circumstances, it is felt that the procedure carries little if any risk, while offering valuable information as:

1. Proof of whether further treatment is necessary.

2. Estimation of the length of time treatment dilatations will be necessary. A complete circumferential burn will have to be dilated over a much longer period of time, if cure is possible, than one which involves only part of the circumference.

3. Value of early bouginage can be more accurately evaluated.

4. Prognosis as to whether dilatations will provide a cure. A deep, ulcerated, cicatricial burn will probably form a stricture in spite of dilatations.

Although diagnosis of stomach damage, resulting from the swallowing of a corrosive, is not included under the subject of this review, possibility of its ocurrence must always be kept in mind. McKibben and Lee[14] warn that pyloric stenosis has not only been reported in patients with esophageal stricture, but has occurred also in patients with no demonstrable esophageal lesion.

A review of the literature back to 1882, by Gray and Holmes[15] in 1948, revealed 139 cases of pyloric stenosis following the ingestion of some corrosive. Although alkalies will exert most of their damaging effect on the esophagus, and acids on the stomach, the swallowing of an alkali can cause severe damage to the stomach. Conversely, acid ingestion can cause esophageal trauma, and in most cases where an acid causes an esophageal stricture, a stricture at the pyloris of the stomach will also occur. As in the esophagus, the severity of stricture at the pyloris will depend on the

extent and depth of penetration. The resulting fibrosis of the gastric mucosa obliterates the gastric glands, altering digestion. Varying degrees of pyloric stenosis will be caused by cicatricial penetrating burns.

TREATMENT.

As in causes of other serious accidents, treatment should begin with prevention. The passage of the "Federal Caustic Act," requiring the use of conspicuous "Poison" labels, which was accomplished by years of work from the Committee on Lye Legislation of the Section on Laryngology of the American Medical Association, has reduced the incidence of lye burns to the esophagus by 90 per cent according to Jackson.[6] The remaining 10 per cent would be greatly reduced if the public were educated to the seriousness of corrosive ingestion so that these preparations would not be brought into the home unless absolutely necessary, and any excess would be promptly discarded or made inaccessible to children. The cases of suicidal intent cannot be reduced, of course, except by restriction of sale of these preparations to the public.

For convenience, management of esophageal burns can be divided into immediate treatment, intermediate therapy, and the treatment of the formed stricture.

Immediate Treatment.

Attempt at neutralization is advocated by most authorities. Administration of vinegar in one-half strength, dilute acetic acid (2 per cent), or orange juice, along with copious amounts of water, is recommended for ingested alkalies. Sodium bicarbonate in water is suggested for neutralizing acids. Kiviranta[16] demonstrated that antidotes have no value unless administered immediately; he found no evidence that attempts to neutralize after the first minute had any effect on the clinical course of the patient, and even taken within one minute it did not prevent injury to the tissues by a corrosive substance. The thought has also been advanced that the chemical action of neutralization might produce enough heat even to increase the burn.

Stomach lavage is contraindicated when the ingested agent is an alkali (Crowe[17]), because of the danger of further damage to the walls of the esophagus and stomach. When the agent is an acid, washing-out of the stomach might be of value in avoiding pyloric stricture.

Administration of olive oil and milk during the first 24 to 48 hours at frequent intervals, is generally accepted treatment of value. They have a soothing influence on the inflamed areas.

Since the patient will frequently refuse to swallow, even his own saliva, during the first two or three days, intravenous fluids may be indicated to prevent dehydration and to provide nourishment. Addition of vitamins *B* and *C* to these parenteral feedings may promote healing.

A bland soft or liquid diet can be given as soon as the patient will swallow, except in the very severe burns where perforation of the esophagus may be suspected.

Adequate antibiotic therapy should be started at once in the form of penicillin and streptomycin, or the "broad spectrum" variety. It was demonstrated by Bosher, Burford, and Ackerman,[18] in their experimentally produced lye burns of the esophagi of dogs, that there was evidence of bacterial invasion of the muscularis varying from actual phlegmon formation to localized intramural abscesses, within 48 hours; therefore, the amount of eventual scarring, and the danger of complications from immediate perforation, will be greatly lessened with the early institution of vigorous antibiotic therapy.

Cortisone administration during the acute phase has several enthusiatic supporters. Many experiments have been carried out on animals which demonstrate the potent inhibitory influence of Cortisone on the inflammatory response of tissue to injury. Spain, Molomert, and Haber,[19] in mice experiments, found scant collections of fibroblasts, insignificant collagen formation, and no new capillaries, if Cortisone was started within the first few hours of injury; however, there

was no difference in the amount of granulations if Cortisone administration was delayed over 48 hours after the injury.

Rosenberg and associates[20] reported that large doses of Cortisone resulted in a remarkable decrease in the tendency towards fibrous stricture formation in lye burns of the rabbit's esophagus, but there was spontaneous perforation of the esophagus in five of the 44 rabbits under Cortisone, which they attributed to the antianabolic, antifibroplastic effects retarding the laying down of a granulation tissue barrier. These experiments are paralleled by reported cases of exacerbation and perforation of existing peptic ulcers during treatment with Cortisone. Weisskopf,[21] carrying out similar experiments on dogs, reported the formation of no strictures in the group treated with Cortisone, whereas all of the control animals living over 35 days developed strictures. Eleven patients with presumptive evidence of esophageal lye burns (mouth burns and dyphagia) were given steroid therapy by Ray and Morgan.[22] Of these, ten patients did not develop strictures, and one formed a clinical stricture after three months; the one failure was attributed to the Cortisone's not having been started until four days after the lye ingestion.

From the preceding investigations, it is apparent that Cortisone therapy, if started within the second day of the accident, will greatly reduce the number of strictures resulting from ingested lye, but a note of caution must be inserted regarding the possibility of its use promoting a greater risk of esophageal perforation and mediastinal infections.

The swallowing of a string, as soon as possible, during the acute phase is recommended by most authorities. This helps to prevent complete atresia in severe cicatricial burns, and provides a guide for later endoscopic examination and dilation. The use of an indwelling feeding tube is unwise, because of the danger of pulmonary complications and because of its acting as a foreign body to stimulate greater formation of granulations. Daly[13] also suggests that it may delay the passage of food with the result that food particles may become enmeshed in the granulations, also causing foreign body reaction.

Intermediate Therapy.

Since the burned esophagus tends to start contraction during the third week, prevention of a stricture becomes the main concern during this latent phase. Supportive measures should be continued as outlined under immediate treatment. In addition, the advisability of dilating the damaged esophagus, as soon as the patient is able to swallow, must be considered.

Salzer[23] is credited with being the first to advocate early dilatations. In 1920 he reported 12 cases in which dilatations had been started on the second to sixth day after ingestion of the corrosive.

Attention was drawn to Salzer's report by Bokay,[24] in 1924, when he reported the use of early dilatations in 132 patients with excellent results in 117. Good results in 90 per cent of 180 patients was reported in 1934 by Salzer[25] with his method of early dilatations.

In this country, Martin and Arena[26] reviewed the subject of lye ingestion and reported three cases of early dilatation in 1939.

Early dilatations were begun, in 1924, by Gellis and Holt[27] in the Harriet Lane Home of the Johns Hopkins Hospital. They reported on 110 children with a history of lye ingestion in 1942. Dilatations in 41 of these patients were started immediately if there was not great swelling of the fauces, and it was never postponed more than four days. Bougies consisting of soft rubber catheters filled with lead shot were used from No. 14 to 34 French. Dilatations were started with a small size bougie and worked up until the largest size passed with difficulty. No force was used in passing the bougie, and if evidence of bleeding appeared the treatment was discontinued for the day. The amount of treatment required was found to vary with each patient; however, the usual schedule consisted of daily dilatations for two weeks, three times a week for the next two weeks, twice a week for two months, once a month for six months, and two or three times a year for two or three years.

If any increasing difficulty was encountered during the

course of treatment, daily dilatations were again resumed. Each treatment was begun with a bougie two sizes smaller than the largest used in the preceding treatment. The largest bougie used in each treatment was left in place for as long as possible up to 30 minutes. Of the 41 patients treated by this method, only one developed a stricture; conversely, of 14 patients of their series who received no dilatations, six formed strictures. The safety of this method was demonstrated by no accidents or deaths occurring in the 41 patients so treated.

The rationale for treatment of esophageal burns by early dilatations is based on the supposition that contraction of the collagen fibers laid down in the damaged area, is prevented. This form of treatment has also been championed by Crowe,[17] Kernodle,[28] and Holinger.[29]

Other authors disagree with the management of esophageal burns by early dilatations. This is based on the belief that the esophagus should be put at complete rest during the inflamed period, and that early dilatation serves only to maintain the inflammatory state. Stumboff[12] recommends direct bouginage through the esophagoscope when the stricture starts to form on the third to the sixth week. Dilatations were started by Vinson[20] only after the lumen had contracted by scar tissue to a size that would not permit the free passage of fluids; his report included a high percentage of deaths, revealing the danger of vigorous dilatation of a contracted scar.

Belief that dilatations of any kind are useless, was expressed by Burford, et al.,[31] who felt that modern esophageal surgery had outmoded this form of treatment. They maintained that all established strictures of the esophagus were surgical problems.

Late Treatment.

When a cicatricial stricture has formed, weighted bougies of the type used in early dilatations are not effective, although they may still be of value in the semi-lunar or "shelf type" of narrowing.

Blind bouginage, other than with the mercury or lead-filled

type has long been condemned, because of the danger of hemorrhage, production of false passages, or perforation with mediastinitis. Before other methods were available, Tucker[32] quotes Trousseau: "Sooner or later, all cases of stricture of the esophagus die of the bougie."

If dilatation of the established stricture is considered to have a chance of success, the acceptable methods include:

1. Direct dilatation through the esophagoscope, using Jackson dilators. This method can be used in cases of a single stricture, but it is hazardous in the presence of multiple strictures.

2. Olive-tipped bougies passed over a previously swallowed string eliminates the danger of the bougie entering a blind pouch. This method does not require gastrostomy, and simplifies the task of the surgeon if gastroesophageal anastomosis is to be done later.

3. Retrograde bouginage by the continuous string method of Tucker[32] in gastrostomized patients, is the safest method for extensive or multiple strictures.

4. Pneumatic bougies placed under fluoroscopic control.

It is generally accepted that dilatation of esophageal strictures should be continued until it becomes apparent that a functional esophagus cannot be obtained, or until it appears that further attempts at bouginage are dangerous. Only then should a resort to surgical methods of repair be considered.

The indications for abandoning dilatations and resorting to surgery are:

1. Impermeable strictures where there is a complete stenosis and attempts to establish a lumen have failed.

2. Scarring that has resulted in pocketing and marked irregularity or fistulas.

3. Persistent poor response to dilatations where the lumen cannot be maintained at a functional size.

4. Persistent ulceration for many weeks, as evidenced by blood streaking on the bougie.

5. When the scarred esophagus cannot be dilated without danger of periesophagitis and mediastinitus.

6. When cooperation of the patient cannot be obtained for the necessary regular dilatations because of a psychotic personality.

Surgery of the esophagus has made great advances during the past 20 years, due to improved surgical techniques, greater knowledge of physiology concerning fluid and electrolyte balance, the great advances in anesthesiology, and the introduction of chemo- and antibiotic therapy.

Many techniques have been developed for restoration of the normal swallowing mechanism in the damaged esophagus which cannot be rehabilitated by bouginage. These methods can be conveniently divided into the antethoracic and intrathoracic approach.

Antethoracic.

In 1913 Torek[33] reported the first successful case of resection of the thoracic esophagus for carcinoma. He brought the cervical stump of the esophagus out to the skin surface. A long rubber tube was fixed in the gastrostomy opening, and the patient placed the other end into the cervical esophageal opening when he wished to swallow.

A skin-lined tube connecting the cervical esophageal fistula with the gastrostomy opening was done on six patients by Yudin[34] in his report of 80 cases of surgical construction of the artificial esophagus. He claimed a good functional result in these patients that survived, but one patient died between procedures, and two developed fatal peptic ulcers in the skin of the new esophagus. This complication, plus the necessity of many surgical stages and prolonged hospitalization with a distasteful cosmetic result, has limited the use of this procedure.

Subcutaneous jejunal substitution was used by Yudin in most of his cases, in conjunction with a cutaneous tube if

the jejunum would not reach. He feels that this is the procedure of choice, and he is joined in this belief by Finnerty;[35] however, the esthetic result of this procedure is also subject to criticism.

Most surgeons at the present time feel that the antethoracic approach is obsolete in comparison with the improved methods of intrathoracic substitution.

Intrathorac.

Resection of the scarred area, and end-to-end anastomosis of the esophagus, can be done for the single short stricture of less than five cm. Gross[36] in 1948 reported one successful case treated in this manner. This type of repair is not frequently applicable, because cases severe enough to warrant surgical intervention usually have multiple strictures.

Esophagogastrostomy, with or without resection of the damaged esophagus, has been very popular with many surgeons. Biondi[37] is credited with doing the first studies for this procedure in 1895, but he reported difficulty with leakage, and frequent recurrence of stricture formation at the site of the anastomosis. In their series of esophagogastrostomy in 52 dogs, Carter, et al.,[38] reported that no strictures formed if the end-to-side type of anastomosis was made, where one end of the esophagus was anastomosed either to the edges of the incision in the fundus of the stomach, or to the edge of a circular area excised from the fundus; they also reported an 89 per cent incidence of perforation at the line of anastomosis when no anchoring sutures were placed in the stomach or esophagus, and no incidence of perforations when both the stomach and esophagus were anchored in position.

The first case of esophagogastrostomy found by this author, was that reported in 1935 by Decker,[39] done for carcinoma. This procedure was championed by Sweet[40] in 1946, in his report of three successful cases done for esophageal stricture in which he states that, "in cases of intractable stricture of the esophagus resulting from chemical burns, an esophagectomy followed by a high intrathoracic esophagogastric anastomosis is superior anatomically, physiologically and psycho-

logically, to any form of external esophagoplasty as a method of restoring functional continuity of the upper alimentary tract."

Esophagogastric anastomosis may be the procedure of choice in strictures of the lower third of the esophagus. As the level of the anastomosis goes beyond the level of the aortic arch, greater difficulty is encountered, especially with a previously gastrostomized scarred stomach, and the risk of an anastomotic leak is greatly increased.

Fatti, et al.,[41] reported 40 cases of esophagogastrostomy, all of which were anastomosed above the aortic arch. There were 24 patients in whom restitution was complete, eight who required occasional bouginage, and eight who died after operation, a 20 per cent mortality. They attributed the unsatisfactory results to the anastomosis being made at, or below the site of esophageal fibrosis. The deaths occurred from peptic esophagitis and gastric ulceration with perforation.

Intrathoracic esophagojejunostomy has been advocated by some authors for lesions involving the upper third of the esophagus. Reinhoff[42] described this method of mobilization of the jejunum and anastomosis with the esophagus in 1946. The main disadvantage of this procedure is that the stomach, duodenum and upper jejunum are side-tracked by the food, and serve only to secrete juices into the intestinal tract.

More recently, the ascending and transverse colon have been used in esophagocologastrostomy or pharyngocologastrostomy, and two cases were reported by Nardi[43] this year for repair of extensive lye strictures without resection of the esophagus. Mahoney and Sherman[44] reported one case of total esophagoplasty, using intrathoracic right colon for carcinoma in 1954. This procedure has the advantage of the colon providing enough length so that the upper anastomosis can be made at the most optimal site, and all of the scarred area can be by-passed. The colon also seems to have a higher resistance to the acid-peptic secretions of the stomach, as demonstrated by Dragstedt and Vaughan,[45] who found that a loop of colon transplanted to the stomach of a dog was

normal both macroscopically and microscopically after one year.

It may not be practical to remove the scarred esophagus if there is considerable periesophageal fibrosis, since the planes are obliterated and severe bleeding may occur. In such a case, the transected ends may be turned in to form a blind pouch. Denton and Bradshaw[46] showed in experiments on nine dogs that the closed-off esophagus becomes a mucous filled cyst which produces no adverse effect. The cyst is self-limited in size, since the pressure developed from within causes atrophy of the mucous glands. If the esophagus is left *in situ* the vagi are not interrupted, and the annoying sequellae of gastric atony and retention are avoided; however, the possibility of carcinomatous development arising from the strictured site still remains. Carver and associates[47] reported two such cases in their analysis of 233 patients with lye strictures, Cecil[48] reported two similar cases, and this author has had one in a patient that had undergone an esophagogastrostomy by the thoracic surgical department.

When resection of the esophagus is done, it has been noted that the more rigorous the bouginage was pursued prior to surgery, the healthier the strictured portion appeared. Cody[11] found cases in which there had been little or no effort at dilatation, to have the most reaction and infection.

ANALYSIS OF CASE STUDY.

In this series of 51 patients, with history of evidence of chemical ingestion, early diagnostic esophagoscopy revealed only eight that needed follow-up treatment.

Four patients were treated by the "Salzer Method," approximately as outlined by Gellis and Holt.[27] One had a complete circumferential burn of the upper third of the esophagus and the other three demonstrated linear burns. All have been kept under observation for over two years, and none have developed signs of esophageal narrowing.

One patient was not started on immediate bouginage, and an esophagogram one month later revealed narrowing in

the upper third with some delay of the barium. Weekly dilatations with olive tipped bougies over a string were started. Barium study of the esophagus 13 months later did not show any abnormality, although bouginage had been discontinued three months previously.

A 16-month-old baby did not receive the advised follow-up treatment and developed a stricture requiring gastrostomy. This patient is now under treatment with weekly retrograde bouginage. She is currently dilated to a size-34 French. This gastrostomy could probably have been avoided if early dilatations had been instituted.

Another patient, who did not have follow-up treatment, became completely obstructed and a gastrostomy was done in another hospital. When he came under our care, he had been fed through the gastrostomy for six months. Numerous attempts to pass a string were unsuccessful. External esophageal exploration was done by the surgical department, and a silk string was inserted, with the ends threaded into the mouth and out the gastrostomy fistula. The patient expired 12 days after this procedure, with post-mortem findings of an esophagopleural fistula, left pyopneumothorax, pulmonary atelectasis, pneumonia, and barium in the lung and mediastinum.

Comment. Early treatment by bouginage might have rehabilitated this patient. When attempts to pass a string failed, surgical reconstruction with one of the intrathoracic methods would have been preferable to the attempt that was made.

The other patient of this series has been discussed previously. He had no ill effects from diagnostic esophagoscopy done nine days after contact with lye; however, an ill-advised esophagoscopy done 27 days after the ingestion resulted in perforation and death in two days.

Comment. This case illustrates the safety of early esophagoscopy and the danger of the same procedure during the period of slough and scar tissue contraction. If this patient had been started on dilatations with the mercury-filled bougies

following the first esophagoscopy, he could probably have been rehabilitated.

It appears evident that the five patients who received adequate peroral bouginage will have a satisfactory result. The three others, who should have had the same treatment, might well have had a similar outcome.

The nine patients who revealed areas of mild erythema by early esophagoscopy, so far have shown no need for further treatment. These, and the 34 others who had no evidence of esophageal damage, escaped the unpleasant ordeal of repeated bouginage, and none have returned with evidence of esophageal narrowing.

CONCLUSIONS.

1. Diagnostic esophagoscopy is a safe procedure in lye burns of the esophagus when done within five to ten days after the ingestion.

2. Every patient with a suspected lye burn of the esophagus should be examined by esophagoscopy during this period to determine presence or absence of a burn.

3. Severity of a burn, approximate length of time treatment will be necessary, and chances of ultimate cure can be determined by this initial esophagoscopy.

4. Esophagoscopy is a less safe procedure during the third and fourth week after the ingestion of lye.

5. Ingested chemicals, other than lye, do not ordinarily penetrate to the extent of causing ultimate stricture formation in the esophagus.

6. Early dilatations, with weighted bougies, is preferable to delaying them until the stricture has formed.

7. Whether the procedure of dilatations is of the early or late variety, it should be given adequate chance of success before the recommendation of surgical intervention is advised.

8. When surgery becomes indicated, the intrathoracic ap-

proaches are the most desirable, and of these, the use of the colon appears to offer the best chance of success.

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404 David Whitney Bldg.

THE COLLEGE OF MEDICAL EVANGELISTS.

A course in "Reconstructive Surgery of the Septum and External Nasal Pyramid" will be sponsored by the Departments of Otolaryngology of the College of Medical Evangelists School of Medicine and the University of Southern California School of Medicine, and will be given at White Memorial Hospital, Los Angeles, Calif. The course will begin at 7:00 P.M., January 6, 1959, and end 12:00 noon Friday, January 16. Dr. Maurice Cottle will be the director. All applications should be forwarded to Leland R. House, M.D., 1720 Brooklyn Avenue, Los Angeles 33, Calif.

TOPICAL ANESTHESIA FOR BRONCHOSCOPY.*

JAMES R. MCKINNEY, M.D.,†

McKinney, Tex.

The advantages and disadvantages of various anesthetic agents for anesthesia in bronchoscopy have been extensively argued. This study is concerned with the employment of a single drug (cocaine) in varying concentrations and by different techniques. We have tried to evaluate as objectively as possible the relative merits of the transtracheal technique of anesthetization as compared with a spray or drop instillation of the agent. In the first of these the anesthetic is introduced into the tracheal lumen by a syringe and needle passed through the skin of the neck and the crico-thyroid membrane. In the spray and drop method the pharynx is sprayed, using an atomizer and 5 to 10 minutes later, a laryngeal mirror is introduced and anesthetic solution dripped through the glottis into the larynx and trachea.

The evaluation is based upon three criteria:

1. Safety—which implies avoidance of systemic reaction to the drug and accidents inherent to the technique itself. It is recognized that various drugs have relative degrees of safety in this respect, but both the total amounts in milligrams and the concentrations of cocaine used in this study are considered to be within acceptable limits.

2. Objective Evaluation by Endoscopist. Anesthesia must be adequate to permit the introduction of the bronchoscope without causing discomfort or provoking troublesome swal-

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*This study was carried out under the direction of the late Dr. James Carswell, Jr., Chief of Thoracic Surgery at the V. A. Hospital, McKinney, Texas. The bronchoscopic procedures were performed by Drs. Milton Hoffman and William Viar of that section, and the anesthesias were administered by Drs. Frank Davidson and James McKinney of the Section of Anesthesiology. Observations of the factors chosen to satisfy the criteria were made as a joint effort by two or more of these physicians in each case. The data were collected by Drs. Davidson and McKinney. The statical significance of the data was determined by Mr. John Geers, Clinical Psychologist, attached to the Section of Anesthesiology.

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lowing, gagging, or cough. It should permit manipulation of the instrument at the carina and in the bronchi, and passage of suction tip and biopsy forceps without stimulation of reflex coughing. To obviate personal bias the endoscopists in this study were unaware of the method of anesthetization employed.

3. Subjective Evaluation by the Patient. The induction of anesthesia should be accomplished with a minimum of discomfort to the patient because of compromising positions, distasteful solution in the mouth, etc. The anesthesia should be sufficiently complete, that the actual operation causes neither pain nor discomfort. In addition to pain or discomfort, freedom from a degree of reaction to the anesthesia characterized by apprehension, a feeling of ill-being, palpitation, increased perspiration, and faintness are considered significant in influencing the patient's interpretation of the satisfactoriness of the anesthesia.

METHOD.

Observations were made upon 67 consecutive diagnostic bronchoscopic examinations. Premedication consisted of phenobarbital, 0.1 h.s., the evening before operation, and seconal, 0.1, and morphine sulfate, 0.016, by hypo approximately one hour before operation. The patients were anesthetized in a room adjoining the bronchoscopy clinic, and care was taken that the bronchoscopist did not ascertain the method used. The two methods were varied indiscriminately. The spray-drop method was used in 29 cases and the transtracheal in 36. In the transtracheal group 3 cc. of 5 per cent cocaine were introduced in 21 cases, and the same amount of 10 per cent cocaine in 15 cases. Two cases were eliminated from the scoring because bronchoscopy was not completed, due to anatomical difficulties in introducing the instrument at the time of the investigation.

EVALUATION.

Results were observed and tabulated at the time of introduction of the scope, and again during manipulation of the intrument deep within the trachea or bronchi. Each case was scored, using the arbitrary standards of 5 for excellent,

4 for good, 3 for fair, 2 for poor, and 1 for unsatisfactory, for each of these factors—swallowing, gagging, coughing, general comfort and general behavior during introduction of the bronchoscope. Thus a perfect score for introduction was tabulated 5, 5, 5, 5, 5 with a total of 25; entirely unsatisfactory anesthesia for introduction resulted in a score of 1, 1, 1, 1, 1 with a total of 5.

A second score was compiled for satisfactory results during manipulation of the instruments in the trachea, and based on the factors of coughing and general comfort and behavior. These were similarly scored 5, 5, 5 for a total of 15 for entirely satisfactory, and 1, 1, 1 for a total of 3, if quite unsatisfactory.

A summary of the scores follows:

SUMMARY OF TRANSTRACHEAL VERSUS SPRAY TECHNIQUE.

SPRAY			2 cc. 5%			3 cc. 10%	
Case	Introduction	Bronchoscopy	Case	Introduction	Bronchoscopy	Case	Introduction	Bronchoscopy
1	20	7	2	25	14	25	24	14
3	25	14	4	25	15	27	22	14
5	24	13	6	24	14	35	22	14
8	25	12	7	24	12	36	25	12
11	16	10	9	25	13	40	25	13
12	25	12	10	24	14	42	25	15
14	22	9	13	22	9	43	25	13
15	25	12	16	25	13	45	20	15
23	25	14	17	25	15	47	24	15
24	25	14	18	23	13	49	22	15
26	17	12	19	25	12	51	25	15
28	14	9	20	19	14	55	25	15
29	17	10	21	25	15	53	25	15
30	23	14	22	23	12	57	25	15
31	22	12	61	25	15	60	25	15
32	22	12	62	25	15			
33	25	12	63	25	15			
34	25	14	64	25	15			
37	21	12	65	25	15			
39	24	14	66	25	13			
41	25	11	67	24	15			
44	12	8						
46	25	14						
48	9	3						
52	25	15						
54	25	15						
56	19	15						
58	25	11						
59	25	15						

Cases 38 and 50 were discarded because of failure to introduce the bronchoscope for anatomical reasons.

STATISTICAL ANALYSIS OF RESULTS.

Introduction	Bronchoscopy
Spray (N = 29)	
M = 21.79	M = 11.90
S = 4.43	S = 2.70
Sm = .84	Sm = .51
Transtracheal 5 per cent (N = 22)	
M = 24.23	M = 13.77
S = 1.28	S = 1.53
Sm = .30	Sm = .33
Transtracheal 10 per cent (N = 15)	
M = 23.93	M = 14.33
S = 1.62	S = .99
Sm = .44	Sm = .26
Transtracheal (5 per cent + 10 per cent) (N = 37)	
M = 24.11	M = 14.00
S = 1.45	S = 1.33
Sm = .24	Sm = .22

"t" Tests of Significance of Difference in Means

5% transtracheal introduction	M = 24.33	
Spray introduction	M = 21.79	
t = 2.77 d.f. = 49		Significant beyond .01 level
5% transtracheal bronchoscopy	M = 13.77	
Spray bronchoscopy	M = 11.90	
t = 3.07 d.f. = 49		Significant beyond .01 level
10% transtracheal introduction	M = 23.93	
Spray introduction	M = 21.79	
t = 2.25 d.f. = 42		Significant beyond .05 level
10% transtracheal bronchoscopy	M = 14.33	
Spray bronchoscopy	M = 11.90	
t = 4.26 d.f. = 42		Significant beyond .01 level
Combined transtracheal introduction	M = 24.11	
Spray introduction	M = 21.79	
t = 2.64 d.f. = 64		Significant beyond .02 level
Combined transtracheal bronchoscopy	M = 14.00	
Spray bronchoscopy	M = 11.90	
t = 3.75 d.f. = 64		Significant beyond .01 level
Transtracheal 5% introduction	M = 24.23	
Transtracheal 10% introduction	M = 23.93	
t = .57 d.f. = 35		Not significant
Transtracheal 5% bronchoscopy	M = 13.77	
Transtracheal 10% bronochoscopy	M = 14.33	
t = 1.33 d.f. = 35		Not significant

INTERPRETATION OF SIGNIFICANCE OF RESULTS.

On the basis of this study it appears that the transtracheal technique is significantly superior to the spray and instillation technique for bronchoscopy, insofar as introduction of the in-

strument is concerned, and also for actual manipulation within the trachea and bronchi.

The relative ease with which transtracheal anesthesia can be accomplished, and the rapidity with which anesthesia develops are further factors which, while outside the scope of this investigation, lend significant support to the conclusion of its superiority.

It is of interest that the substitution of 10 per cent for 5 per cent cocaine in the transtracheal technique did not significantly improve the anesthesia.

Since this investigation was carried out the technique has been modified to permit the use of smaller quantities of solution for the procedure, and in most cases it has been found that 2 cc. of solution are adequate.

INTERNATIONAL SYMPOSIUM ON RADIOLOGY IN OTOLARYNGOLOGY.

On December 7 and 8, 1958, the Gruppo Otorinolaryngologica of the Alta Italia will sponsor an International Symposium on Radiology in Otolaryngology under the direction of Prof. Luigi Pietrantoni, Direttore della Clinica dell' Universita de Milan; Prof. Arduimo Ratti, Direttore dell' latitudo di Radiologica dell' Universita de Milan, and Honorary President Prof. Felice Perussia. For further information write the Secretary of the Symposium, Clinica Otorinolaryngologica dell' Universite di Milan, via Commenda 16, Italy.

COMBINED NUTRITIONAL AND PSYCHOSOMATIC MANAGEMENT OF MUCOSAL DISEASE.

The Stress Concept Applied to the Treatment of Ear, Nose, and Throat Diseases.*

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Pasadena, Calif.

Dietary supplementation as part of the treatment of ear, nose and throat diseases has been so widely discussed in the literature that the subject seems well-nigh exhausted. Convincing results were obtained with various dietary regimens and by the prescription of an appropriate vitamin therapy. Clinical experience of this kind has been confirmed, not only by animal experiments but also by controlled tests in human volunteers; thus a number of deficiency diseases were recognized and standard treatment methods developed, but beyond these pathologic conditions there is a wide range of partial or potential deficiencies with ill-defined and variable signs and symptoms which may or may not respond to a change of diet or to vitamin therapy.

Partial deficiencies or borderline states are by their very nature difficult to recognize. Each case is different and occurs in an individual setting of physiologic and environmental conditions; thus generalizations are of doubtful value and little can be expected from use of the statistical method. Animal experiments are at best suggestive, and the same holds true with regard to tests in volunteers or group observations. Small wonder, then, that the field is rife with controversy and abounds with subjective statements from opposing camps. Under these circumstances not much can be expected from a mere review of past observations, and the only hope for clarification of the issue lies in a fresh approach

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to the problem of dietary deficiencies and nutritional supplementation.

We might begin by considering the basic difference between pathology (morphology) and biochemistry, as pointed out by Williams.[21] He remarked that the pathologist is often unable to distinguish morphologically between healthy cells and those which are deficient in some essential principle. Pathology is limited to the study of end-products of disease processes, while the field of biochemistry is much larger, concerned as it is with all phases of life—not only the destructive ones. This distinction is of greatest importance in modern therapy which is no longer restricted to the treatment of symptoms, but also aims at improving the patient's general health by attention to the underlying factors.

What is health? If we consult the dictionaries we do not receive any real answer. Webster defines health as "the state of being hale or sound in body, mind, or soul; especially freedom from physical disease or pain." Essentially the same information comes from medical dictionaries. Blakiston, for instance, tells us that health is "that condition of bodily soundness in which the functions are performed normally; a state of well being and freedom from disease." The reference to "normal" functions is not much of a lead either. Ivy,[8] in a very thoughtful paper on "What is Normal or Normality?" arrives at a description of the physiologically normal condition of the body "as that relatively steady state which results from well integrated functions supported by margins of safety in materials, structures and processes," but he adds immediately that this is not "a definite reference point from which to measure or with which to make a comparison. Provocative as this physiological statement may be, it is certainly too vague to serve as a directive in the selection of therapy, and leaves us with only a general notion of what might be achieved.

At this point the stress concept, as proposed by Selye, comes to our aid. His definition gives the term "stress" a specific meaning:[14] it is an attempt to arrive at a unified basis for various disease processes and their signs and symptoms. Others before had tried such a unified approach, since

the days of Hippocrates,[15] but it was Selye's contribution that he based his all-encompassing concept on findings in the field of hormonal interaction and tested it by exact laboratory experiments.

This new approach has greatly stimulated research as well as the treatment of various diseases. The stress concept was taken up by different medical specialties, foremost among them endocrinology, with psychosomatics running a close second; but there is no branch of medicine which has not profited by the discovery of stressors and the evaluation of stress-producing situations. With ever more sensitive techniques of investigation the number of stress-producing agents and situations is constantly increasing.

It would, however, seem that in paying attention to specialized applications of the stress concept Selye's basic idea of a unified theory of disease has been lost sight of. In the light of his original plan the most remarkable discoveries in the field of hormonal interaction, or in any other of the specialties, are only means of clarifying the common basis of health and disease. A frame of reference for hitherto unrelated biological events has been established. This is important for the recognition of pathologic processes, but its significance does not end there. It is only one step from a unified concept of disease to a unified concept of therapy. By improving several seemingly unrelated functions we come closer to the ultimate objective of all medicine—to promote health. The coordination of as yet separate methods or procedures into an integrated plan of treatment holds out promise of a much greater and more lasting therapeutic effect.

Research and therapeutic experience have progressed far enough for such a unified approach to be applied in the management of common ear, nose, and throat diseases. This can be best demonstrated with regard to the status of the mucous membranes of the ear and the respiratory passages, in health and disease.

Nobody will underestimate the value of topical treatment, particularly chemo- and antibiotic therapy, in combating carriers of infection; but still more important are the natural

defenses of the mucous membrane, or to put it differently, its ability to deal with noxious agents of any kind. It has been convincingly shown that resistance to infection is lowered in stressful life situations.[7] Similarly endocrine and autonomic disturbances result in impairment of the protective functions of the mucous membrane.[6] Nutritional deficiencies not only interfere with smooth recovery from disease, but also change the microscopic structure of the mucous membrane and lower its ability to resist the inroads of stress.[1]

Therapy along any one of these lines at times produces spectacular results. Topical treatment is likely to afford immediate relief; endocrine therapy often leads to remarkable improvement; psychotherapy in one form or another may resolve the conflict-laden situation and thereby remove psychic stress; better nutrition not only increases all-around health but has also been found effective in lowering the incidence of upper respiratory disease.[10] The most promising approach, however, would seem to be a combination of these two last-named treatment methods with topical therapy to alleviate the immediate complaint.[4] It will be shown that dietary supplementation and psychotherapy are mutually supportive, one potentiating the beneficial effects of the other.[13,17,18]

Such an integrated plan of therapy amounts to a practical application of Selye's original concept of stress. The various stressors and stress-producing situations are interlinked, and by attacking several of them simultaneously it is possible to influence the overall reaction pattern of the patient. The whole is more than the sum-total of its parts.

Examples of how stressors and stress-producing situations are interlinked might easily be found in all fields of medicine and by critical evaluation of any therapeutic approach. Concentrating on dietary supplementation, there can be no doubt that this measure by itself is often highly effective.[2,3] Recently Watson, of W. C. Kalash Laboratories, Pasadena, Calif.,[19] obtained significant relief of mental symptoms by inclusive dietary supplementation; but dietary intake represents only one aspect in a much more comprehensive setting of internal

and external conditions which determine the degree of well-being of the human organism. This has been well expressed by Dann and Darby;[5] after surveying the many experiments and clinical observations regarding dietary deficiencies, they came to the conclusion that "the introduction and universal consumption of an ideal diet would not ensure abounding health; it would lay only one of the foundation stones on which such health might be based. A multitude of environmental factors, including as not the least important, the individual's reaction to his fellow members in human society, must be in correct adjustment before such health can ensue."

That diet should be evaluated in a setting of numerous conditions affecting the external and internal environment has been still more impressively pointed out by Kruse.[11] A clear distinction is made between "nutrition" as a bodily process and "diet," which is defined as a regimen of food supporting the bodily processes of nutrition. At first this may appear quite arbitrary, but we must remember that an increase of dietary intake is routinely prescribed under such conditions of special stress as growth, pregnancy, and hard labor.

The recogntion of multiple stress-producing factors has not remained without impact on the treatment of nutritional disturbances. Jolliffe,[9] for instance, states that the successful treatment of vitamin deficiencies "involves treatment, and whenever possible, the elimination of the stress or conditioning factors. It involves symptomatic treatment, for a comfortable patient is of great aid to recovery. It finally involves nutritional therapy in sufficient amounts over a sufficient period of time to evoke maximal reversal of the nutritional syndrome."

A similar plan for a unified management of diseases involving the mucosa of ear and respiratory passages has proved very successful during the past few years. While a detailed report is reserved for a later communication, a few of the underlying principles will be discussed in the following:

According to Selye[16] "the primary object of the stress reaction is the maintenance of life and the re-establishment of normalcy during and following damage or functional activity."

Topical treatment, if necessary, combined with systemic therapy will as a rule result in repair of damage to the mucosa and re-establishment of its physiologic functions. This constitutes adequate management as long as we consider diseases of the mucosa of ear and respiratory passages as merely incidental, somewhat on the order of an unfortunate accident; but such a purely mechanistic view, attributing mucosal disease entirely to a foreign invader or to some kind of extraneous cause is no longer tenable. Mucosal disease occurs as a result of disproportion between insult and ability to deal with it. Merely topical treatment, even if combined with systemic support of local defense, leaves the underlying condition of failing resistance unchanged. The presenting acute illness is actually an exacerbation of a chronic condition of lowered physiologic responses, and it is due to this weakening of natural defenses that stressors which otherwise might have remained innocuous are able to produce disease.

As long as management is restricted to dealing with the acute episode, the patient is left in the same condition of disease proneness as before—we have ministered to disease but not to health; thus stress is regarded as an exclusively negative factor, or as Whitehorn[20] puts it, "stress has become a bad word." In a provocative paper on "The Healthful Benefits of Stress" he remarks that "perspectives have become so distorted at times that health efforts are aimed too sweepingly at the elimination of stress or anxiety." This represents the observation of a psychiatrist, but Whitehorn's advice that "doctors should think more discriminatingly about stress" applies equally to other branches of medicine.

What does all this mean in terms of the treatment of patients with diseases of the mucosa of ear and respiratory passages? Chemo- and antibiotic therapy is applied, and pain relieving drugs are administered; but management is not limited to these emergency measures, and a long-range program to build up the patient's resistance is immediately initiated. Of course, this is not possible in all cases, as many patients look upon their doctor simply as a trouble shooter; they take off a few minutes from the job, rushing into the office and rushing out again, expecting immediate relief and

perhaps a pep talk, but are not open to further advice. Many others, however, have begun to wonder why they catch cold so frequently or why they are bothered by recurrent attacks of ear, nose, and throat diseases. Patients of this type are not satisfied with temporary relief, and expect the physician to do something about their disease proneness. If approached with tact and sympathy they will cooperate in a long-range medical program. Here the suggestion of a change of diet or dietary supplementation will fall on fertile soil, and the patient has no difficulty in understanding that such measures must be continued for some time before tangible results can be expected; but to suggest dietary therapy without having first gained the patient's confidence will rarely prove successful.

By offering psychological support and prescribing dietary supplementation, an attempt is made to utilize the episode of acute disease as a starting point for improving the natural resistance of the body.[12] At least potentially a new trend has been set up, a spiral toward health replacing the old spiral toward disease.

Selye has drawn attention to the fact that any disease is a crisis involving the entire system. Still more far-reaching is his distinction between various types of adaptation to threatening stimuli. Even in adapting to a state of lowered resistance the human organism exhibits its fundamental tendency toward self-preservation. The unified approach to therapy, combining topical, systemic, psychologic and dietary management is designed to improve adaptation to every kind of stimulus, with the ultimate aim of increasing overall fitness.

SUMMARY.

Re-study of clinical and experimental data in the light of Selye's stress concept has resulted in a new plan for the management of mucosal disease, particularly of ear and respiratory passages. Multiple stress-producing factors call for a unified approach to therapy. Topical and systemic medication is administered to afford immediate relief, but at the same time long-term management, combining psychologic

support and dietary supplementation, is instituted to increase the patient's resistance to disease.

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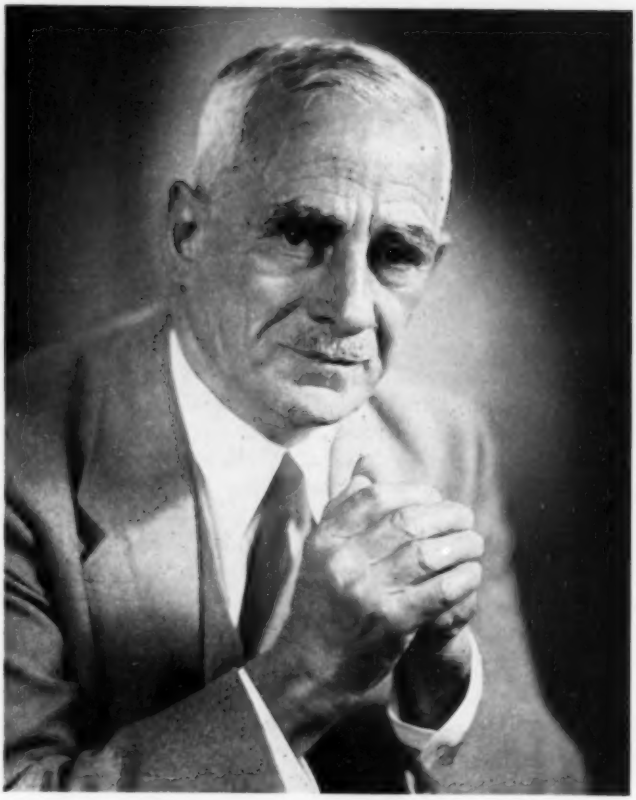
65 North Madison Avenue.

IN MEMORIAM.



—Photo by Ashley and Crippen.

D. E. STAUNTON WISHART, M.D.,

1888-1958.

Dr. D. E. Staunton Wishart died in Toronto in April, 1958, at the age of 69. He had been in ill-health for over a year, but carried on his work until a few months before his death.

Dr. Wishart was the son of Dr. Gibb Wishart, who was a former professor of otolaryngology in the University of Toronto, so he was steeped in the traditions of medicine and the specialty in which he spent his medical career. Educated at St. Andrew's College and the University of Toronto, he received a B.A. degree from University College in 1909, and an M.B. from the Faculty of Medicine in 1915. He was an honor student throughout his medical school.

During the first World War, Dr. Wishart served in the Canadian Army Medical Corps and transferred to the Royal Army Medical Corps later. He saw service in Gallipoli, Serbia, Macedonia and Palestine and was mentioned in dispatches at Salonika in 1917. On returning from overseas he took up the study of Otolaryngology and did post-graduate work in Boston, Philadelphia and Edinburgh, and on his return to Toronto was appointed to the staff of the Hospital for Sick Children. He became chief of otolaryngology at the hospital and an assistant professor in the University, both of which positions he held until his retirement to private practice in 1953. He remained a graduate lecturer in the University.

In 1939 Dr. Wishart was appointed to the editorial staff of THE LARYNGOSCOPE and was assigned the subject of Rhinology in Children. His annual reviews of the available literature on this subject were masterpieces. He had an uncanny ability to get at the gist of a paper, and to cover it briefly, but thoroughly, in his Review.

Dr. Wishart was a member of the American Otological Society and was one of the few Canadians to become its President, which honor was conferred upon him in 1954. He was a former President of the Canadian Otolaryngological Society; a member of the American Laryngological Society, and the Royal Society of Medicine, London. He delivered the Birkett Lecture in Montreal, in 1952, an outstanding honor in Canadian otolaryngology.

One of Dr. Wishart's chief interests was the study of deafness, and he did much research in the problems of hard-of-hearing children. He founded the Clinic for the Prevention of Deafness, sponsored by the Atkinson Charitable Foundation at the Hospital for Sick Children, Toronto.

Dr. Wishart's main recreation interests were trout fishing, golf, and his summer home at Georgian Bay among the Thirty Thousand Islands of Ontario. He looked forward each year to his holiday there with his family.

He is survived by his wife, two sons and a daughter.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

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Meeting: Palmer House, Chicago, Ill.

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Meeting: The Homestead, Hot Springs, Va., March 8-9, 1959.

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Annual Meeting: The Homestead Hot Springs, Va., March 10-11-12, 1959. (Mornings only).

Meeting of the Sections:

Eastern: Savoy Plaza, New York, N. Y., January 8-9, 1959.
Council: Savoy Plaza, New York, N. Y., January 10, 1959.
Middle: Park Plaza Hotel, St. Louis, Mo., January 12-13, 1959.
Western: The Ahwahnee Hotel, Yosemite Valley, Calif., January 17-18, 1959.
Southern: Academy of Medicine, Hotel Atlanta Biltmore, Atlanta, Ga., January 23-24, 1959.

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Meeting: Atlantic City, June 8-12, 1959.

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Place: The Homestead, Hot Springs, Va., 1959.

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Secretary: Dr. Robert M. Hansen, 1735 No. Wheeler Ave., Portland, Ore.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, Ill., October, 1958.
Annual Meeting: October, 1958, Chicago, Ill. (Definite time and place to be announced later).

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY.

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Secretary: Dr. Samuel M. Bloom, 123 East 83 St., New York 28, N. Y.
Meeting: November 12, 1958, in conjunction with the Section on Laryngology, Rhinology and Otology of the Medical Society of the County of Kings and Academy of Medicine of Brooklyn, 1313 Bedford Ave., Brooklyn, N. Y.

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Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6, Pa.
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Meetings: Twice every month, first and third Thursday, 8:30 P.M

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Secretary:
Meeting:

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Secretary-Treasurer: Dr. H. Carlton Howard.
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Meeting:

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Secretary of Ophthalmology Section: Dr. Philip D. Shanedling.
Chairman of Otolaryngology Section: Dr. Robert W. Godwin.
Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

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Meeting: Edgewater Gulf Hotel, Edgewater Park, Miss., May 15-16, 1959.

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Meeting:

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Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Seventh Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Miami, Fla., March, 1960.

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Meeting: October 14, 1958, Palmer House, Chicago, Ill.

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Annual Meeting:

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Four Pages	$ 37.25	$ 46.50	$ 61.50	$ 88.75
Eight Pages	51.50	66.25	89.25	127.25
Twelve Pages	65.00	84.25	117.00	175.75
Sixteen Pages	79.00	102.25	129.50	191.00
Twenty Pages	94.00	119.75	160.25	231.50
Twenty-four Pages	106.75	136.00	180.75	261.50
Twenty-eight Pages	115.50	146.75	193.00	277.75
Thirty-two Pages	133.00	163.25	210.75	311.25

Express charges will be paid by consignee.